The New England
Journal of Medicine

VOLUME 212 JANUARY 3, 1935 NUMBER 1

NEW ENGLAND SURGICAL SOCIETY

THE LIMITATIONS OF ENTEROSTOMY AND UNDESIRABLE EFFECTS INCIDENT TO ITS USE*

BY ISAAC M. WEBBER, M.D.†

ALTHOUGH a life-saving measure when opportunely employed, enterostomy is a procedure by no means devoid of the hazard of undesirable effects. Obviously then, the indications for the insertion of a catheter or tube into an obstructed bowel and its maintenance therein should be clear-cut and a proper technique used in its performance if the vital interests of the patient are to be conserved.

The limitations and undesirable effects of enterostomy to be considered in this paper are primarily those pertaining to its use in early postoperative obstruction of the small intestine. The citation of three case histories will serve to illustrate certain aspects of the subject. Blood chemical changes are to be omitted.

Enterostomy as a therapeutic measure in treating intestinal obstruction has been in use for more than three decades. During this period as a result of accumulated knowledge and experience in its application, many divergent opinions have been expressed concerning its efficacy, indications, and limitations; and in some sections of the United States certain eminent surgeons appear to be restricting its field of usefulness. In support of this statement may be mentioned the admirable results of Cheever obtained by abdominal evisceration with operative evacuation of the small intestine. This measure, considered drastic by some of us, was resorted to in an attempt to relieve a group of eight patients considered well-nigh hopeless because of varied and profound toxic manifestations of peritonitis with intestinal stasis or ileus. Yet there was not a single fatality.

As further evidence of the changing viewpoint in the employment of enterostomy, and in sharp contrast to the inherently dangerous and spectacular method just mentioned, may be cited the simple use of suction siphonage applied to an inlying duodenal catheter. This step has been selected by Wangensteen as his only operative procedure in successfully effecting decompression in nine cases wherein the patients suffered from acute uncomplicated obstruction, such as is most commonly exhibited by the formation of an adhesion during convalescence from an abdominal operation.

The judicious use of any method of treating obstructive lesions of the intestine necessitates a working knowledge of the structural changes and perverted functions present in such affections. It should be recognized in the early hours of obstructive manifestations that the seriousness of the situation to a large degree is dependent upon the morbid anatomic condition underlying the occlusion. Differentiation, if possible, should be made of the following:

(1) Simple mechanical obstruction, i.e., the occlusion at a single point, such as is produced by the plugging of the intestinal lumen with a foreign body or that brought on by enterostenosis or a constricting adhesion, the type of obstruction in which the vascular circulation is but little impaired until relatively late and then largely because of overdistention of the bowel.

(2) Strangulation obstruction; namely, the occlusion of a definite loop or segment of intestine, as in strangulated hernia, volvulus, or vascular occlusion in the mesentery, the form of obstruction in which vascular supply is early jeopardized.

(3) Ileus; or obstruction arising from inhibition of intestinal motility incident to a faulty nervous mechanism, such as occurs in some cases of injured spine, or from toxic paralysis of the neuromuscular mechanism of Auerbach in cases of peritonitis and in other infections.

Experimental and clinical evidence suggests that in bowel obstruction a normal mucosa does not permit a substance, lethal in degree, to be absorbed from its content. Overdistention of the bowel, comparatively rapid in onset, appears to impede the egress of venous blood and thus to result in faulty intestinal nutrition, a disturbance which eventuates in mucosal damage, inhibition of peristalsis, and the escape of

*Read at the Annual Meeting of the New England Surgical Society at Burlington, Vermont, September 29, 1934.

†Webber, Isaac M.—Surgeon, Maine General Hospital, St. Barnabas Hospital and Children's Hospital. For record and address of author see "This Week's Issue," page 32.

poisonous material into the blood stream of the individual. After the bowel mucosa has been damaged and the musculature paralyzed, drainage of more than a short segment adjacent to an inserted tube by indirect methods like enterostomy and naso-gastric suction siphonage cannot be reasonably anticipated. In certain instances, if peristalsis is still active, drainage of the small bowel content by such means appears to be sufficient to prevent lethal overdistention of the intestine until automatic release of the obstructive mechanism occurs. Cases of fatal perforation at the level of obstruction after enterostomy have been recorded in the literature. The possibility of such a catastrophe should be considered in the use of suction siphonage. Yet the likelihood of such a mishap seems relatively small when the method is used in the type of case under consideration.

Repeated observations have demonstrated to surgeons conversant with abdominal complications that, in dealing with instances of complete obstruction of the small bowel, the patient's profound feeling of depression has often abated to a marked degree shortly after release of the distention by enterostomy, even though the obstructing mechanism has not been freed at the time of laparotomy; and that, as the automatic reëstablishment of the continuity of the intestine effects itself, the discharge of gas and fluid through the enterostomy tube has often become so greatly diminished that the tube can be clamped without a reoccurrence of symptoms. These observations and the relatively early demonstration by x-ray examination of flatus in the colon previously devoid of gas were apparently factors which led Wangensteen to attempt decompression in such instances by suction siphonage.

Concurrent opinion inclines to favor the proposition that there are two well-established indications for performing enterostomy on some portion of the intestinal tract. Broadly speaking, they are as follows:

(1) For the removal of deleterious material from the obstructed bowel.

(2) For the introduction of nutritive or therapeutic substance into the intestinal canal.

To state more specifically the requirements for enterostomy in bowel obstruction entails the danger of being in error. However, a suggestion of some positive though not absolute indications may be added as follows:

(1) For uncomplicated adhesive obstruction in selected instances when its direct release greatly increases the possibility of spreading infection, such as might result from inadvertently opening into an appendiceal abscess.

(2) As an occasional adjunct to resection of the bowel in late strangulation obstruction when the involved segment is non-viable.

Knowledge obtained from current literature and from personal experience with obstructive intestinal lesions leads me to believe that such conditions can be temporarily improved or entirely relieved by the use of the naso-gastric catheter of Levine, especially when suction siphonage is applied as advocated by Wangensteen, Schmidt, Willard Bartlett, Jr. and by others.

To illustrate the use of suction siphonage in the treatment of intestinal obstruction, I wish to include at this point the history of a specific instance of its use.

CASE 1. A boy, aged sixteen years, underwent operation for appendicitis forty-eight hours after its onset. The inflammatory process was well advanced, yet it had not spread beyond the confines of the appendix. The operation was performed without apparent incident and the abdomen closed without drainage. The first two postoperative days were uneventful. Listlessness and nausea developed to a noticeable degree on the third day. The temperature remained unchanged at 99.8°F. and the pulse at 84 beats a minute.

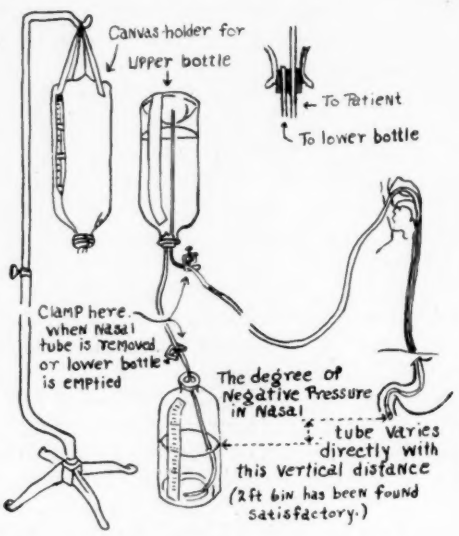

Canvas holder for
Upper bottle
To Patient
To lower bottle
Clamp here when Nasal tube is removed or lower bottle is emptied
The degree of Negative Pressure in Nasal tube varies directly with this vertical distance
(2 ft 6 in has been found satisfactory.)

FIG. 1. Diagram of Wangensteen's apparatus for maintaining suction siphonage.

On the fourth day after operation, I was requested to see the patient because of abdominal distention and vomiting. Examination of the abdomen revealed the distention to be definite but not marked and auscultation determined peristaltic sounds to be feeble and infrequent. On removing the gauze dressing from the abdomen, small bubbles of gas were seen to escape from one of the puncture holes about a silkworm tension suture. The temperature had mounted to 102.2°F. Peritonitis with ileus appeared to be present. A radiogram of the abdomen made without the aid of contrast medium revealed numerous loops of small intestine markedly dilated and the absence of gas in the colon.

On my suggestion, suction siphonage by nasogastric catheter was instituted and maintained (fig. 1). Vomiting ceased and the patient was comparatively comfortable; otherwise, very little change occurred either clinically or roentgenographically during the next four days although the catheter appeared to have entered the duodenum (fig. 2). On

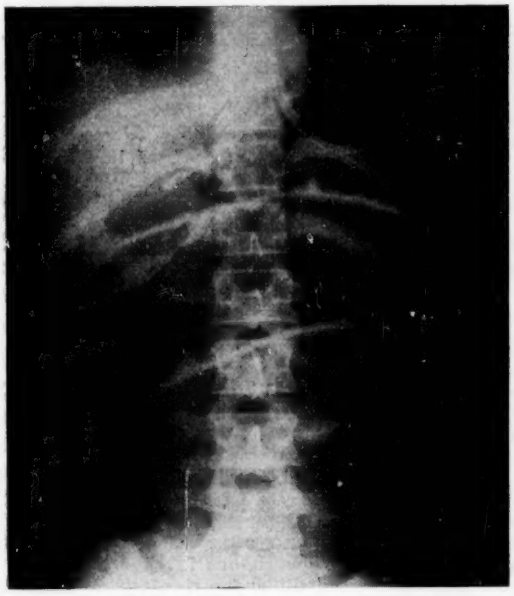


FIG. 2. Radiogram of abdomen in Case 1 made without the aid of contrast medium. Distended loops of small intestine show folds of Kerkring. Practically no gas exists in colon. X-ray picture made four days previously was almost identical.

the sixth day after instituting suction, the patient felt somewhat improved, and five liquid stools appeared during the night. The abdomen was a bit softer. The edges of the skin incision were separated but the deeper structures were intact. X-ray pictures of the abdomen showed definitely less distention of the small gut (fig. 3). Suction applied to the Levine tube was changed to one hour in every four, and the administration of a liquid diet begun. The nasal catheter was removed on the eighth day after its insertion, and progress went on uneventfully with the exception of resuturing the skin.

In regard to the foregoing case, the feeling may be expressed perhaps justly that the employment of suction siphonage, an indirect method of unproved value, for six days without demonstrable evidence of improvement unnecessarily jeopardized the patient's chance of recovery. On the contrary, it must be admitted that in the hands of the average surgeon, a direct attack on such a condition is not devoid of the dangers of shock and the possibility of disseminating toxic materials deadly in character. The mere fact that the patient recovered suggests either that the recovery occurred in spite of the measures employed, or that suction siphonage was of some therapeutic assistance. Other personal observations of its use and a number of successful reports in the literature lend evidence to the latter probability. Certainly, in this particular instance, vomiting remained in abeyance and the patient was made comparatively comfortable; more so, I believe, than if he had been subjected either to jejunostomy or high ileostomy.

In contrast to the foregoing case in which suction siphonage was employed, my second report presents another aspect in which ileostomy was effectively used and seemed at the time the method of choice.

CASE 2. A girl, aged ten years, underwent operation for appendiceal abscess five days after the onset of symptoms referable to the appendix. Entrance to the abscess through an incision just mesial to the right iliac spine was easily made. The necrotic appendix was found lying in the cavity practically free except at its base. The sloughing mass was readily picked up and its base encircled by a ligature preparatory to amputation. The thrombosed vessels of the meso-appendix did not bleed and were not ligated. A drain was placed and

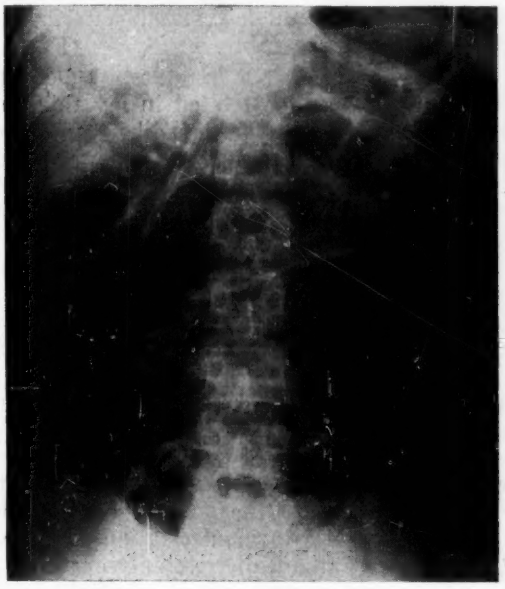


FIG. 3. Radiogram in Case 1 made six days after instituting suction, the first to show less distention of small bowel and appearance of gas in the colon.

a stitch taken to close the mesial end of the incision.

The temperature of 101°F. and pulse rate of 110 a minute fell to normal on the sixth day after draining the abscess. All went well until the twelfth postoperative day when acute, intermittent abdominal pain developed and was accompanied by vomiting. The abscess appeared to be draining satisfactorily to the surface. Auscultation of the abdomen revealed loud, prolonged peristaltic noises synchronous with the height of the pains. A radiogram of the abdomen made without the aid of contrast medium revealed definite dilatation of the small intestine. The general condition of the patient was excellent and a direct attack on the obstructing mechanism was contemplated.

The abdomen was opened through a low midline incision. Residual signs of a general peritonitis were absent. Several loops of lower ileum formed the wall of the abscess cavity which continued to exude pus by the way of the lateral incision. The coils of ileum were so adherent to one another that it seemed well-nigh impossible to separate them

without opening into the bowel or into the pus-containing cavity. Therefore, just cephalad to the most proximal adherent loop, an enterostomy was performed with a No. 16 French catheter according to the method of Witzel. Five days later the patient's bowels moved normally by rectum. On the fourteenth day after enterostomy, twenty-seven days after draining the appendiceal abscess, the patient became ambulatory. There was no appreciable leakage at any time at the site of enterostomy. She was dismissed twenty days after the second operation with both incisions essentially healed, and she has since remained in good health, free from hernia for one and a half years.

Reference in this particular instance has already been made to the fact that, after trying, it seemed practically impossible to free the obstructing mechanism, the adherent loops of ileum, without opening into the bowel or into the pus-containing cavity. Obviously, had such a mishap culminated, the opportunity for a general peritonitis would have been materially increased.

The emergence of such an undesirable effect as fistulous formation, as related in my third case history, is without doubt less likely to become manifest when enterostomy has been performed according to the method of Witzel, and with the catheter protruding from the abdomen through a stab wound somewhat away from the larger incision when conditions warrant. Yet, it must be confessed that a troublesome fistulous complication has developed in one of my own patients subjected to enterostomy, and that I have witnessed a similar crisis in the routine of an associate. When such a fistula is sufficiently near the upper end of the small intestine, unless the ill effects of secretional loss are competently combated, the escape of gastro-intestinal secretions onto the surface of the abdomen may give rise to manifestations extremely painful to the patient, distressingly exacting for the nurse, and productive of physiologic disturbances that may eventuate in death of the patient unless successfully treated.

CASE 3. A girl, aged six years, underwent operation for suppurative appendicitis with peritonitis. Six days later unmistakable evidence of intestinal obstruction became manifest. Pelvic abscess was absent as determined by rectal examination. To relieve this acute situation, enterostomy was performed through a low, left rectus incision. For a short time the patient became more comfortable and vomiting ceased. The tube drained well. On the sixth day after its insertion, the tissues of the abdominal wall and bowel about the enterostomy tube separated, and the tube freed itself. The opening in the bowel was fully three-quarters of an inch long. The outpouring of fluid from the opening constantly bathed the skin of the abdomen. Food taken by mouth likewise made its escape after a few moments. Dressings were changed in rapid succession as gushes of intestinal fluid repeatedly saturated them. In the natural course of events the skin over the greater part of the abdomen became partially digested, weight loss was appalling, and dehydration grievous. The urine, usually passed involuntarily, contained a heavy trace of albumin and numerous red blood cells.

Approximately two weeks after enterostomy had been performed, the patient came under my care. She presented a picture of utter misery. Emaciation was so marked that the ribs stood out in bold relief. Four small decubital or trophic ulcers were present on the skin of the back (fig. 4). The tongue

FIG. 4. View of patient's back showing four decubital ulcers and hideous loss of subcutaneous fat. Almost entire outline of left scapula discernible. Other bony structures protrude.

was dry and the buccilingual membrane in spots was covered with a whitish exudate consistent in appearance with thrush. The greater part of the skin over the abdomen was inflamed, and about the gaping enterostomy incision it had become digested. She was too ill to cry. An attempt to change her position or to administer nourishment met with a brief sickly whine and a grimace at which time, owing to loss of subcutaneous fat, the outline of the orbicularis oris and other facial muscles were plainly discernible. Pus was still being discharged from the appendectomy incision. Evacuation of fecal matter by rectum had not occurred since the operation which was performed two weeks previously. Three blood transfusions had given not more than temporary improvement.

Starvation and dehydration appeared to be the chief obstacles to adequate therapy — yet she refused to take other than a small amount of water. The superficial veins were obliterated by injury from saline infusions and transfusions of blood. The patency of the bowel was determined by injecting saline solution by catheter inserted through the fistula into the lower bowel segment. The administration of a properly constituted liquid diet was then begun by Murphy drip through a Levine tube in naso-gastric position. Normal saline was also given by hypodermoclysis. Gauze dressings were omitted from the abdomen. An elliptical disc two by one inches in its greatest dimensions was fashioned from celluloid. Through a perforation in the center of the disc, the small end of a No. 16 French catheter was inserted. A small safety-pin was placed, like a cotter pin, through the end of the catheter to prevent its withdrawal, and the excess tip of the catheter distal to the pin removed. This oval-shaped disc, with catheter attached, was then slipped into the bowel through the fistula and manipulated into place, so that its position blocked to a very large degree the escape of bowel content. The large end of the catheter was passed through the hole of a similar disc which was superimposed loosely against the raw surface of the gaping incision and held there by another safety-pin (figs. 5 and 6). The partially digested food escaping around the edges of the discs was removed by continuous suction applied to a rubber tube. Stiffness of the rubber tubing thus employed was enhanced by dividing it about one and one-half inches from its lower end and inserting a glass tube about six inches long. The rubber-tipped glass tubing with a small amount of slack was suspended over the

patient from the arc of a hoop supported by the bed. By virtue of the inserted glass tubing, the suction tube largely stood on its end near the partly occluded fistula in the gaping incision. The upper end of the suction tube was kept from toppling by overhead support (fig. 6). The escaping food was

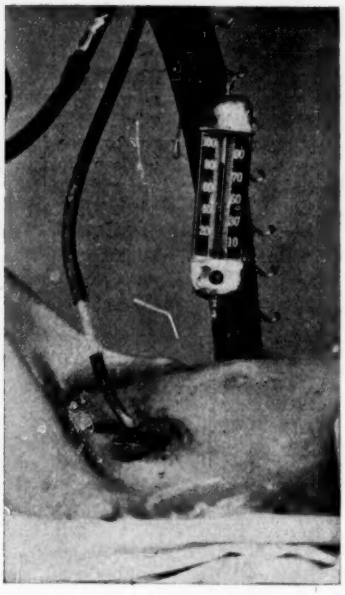


FIG. 5. Photograph of appliance attached to patient's abdomen to diminish loss of food and fluid from fistula, also to permit healing of inflamed skin of abdomen. Over the cecal area the unhealed appendectomy incision is barely visible.

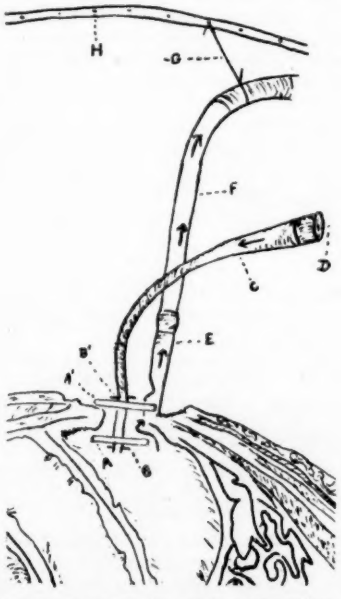


FIG. 6. Diagram of apparatus, and cross section of abdominal wall and underlying gut through the fistula, illustrating figure 5. A, inner elliptical disc; A', outer elliptical celluloid disc; B and B', safety-pins; C, catheter inserted through disc and held therein by safety-pins; D, cork stopper in end of catheter; E, rubber tubing with opening in its side near its lower end; F, section of glass tubing; G, thread supporting suction tube; H, hook in arc of hoop over patient.

thus removed promptly and automatically by suction into an attached bottle. When two or three ounces of intestinal content had been collected by suction they were reintroduced by gravity into the bowel by way of the catheter held in the center of the celluloid discs.

The effectiveness of this mechanical arrangement was attested by the fact that spontaneous, daily evacuations of the rectum promptly occurred after being absent for a period of two weeks. Improve-

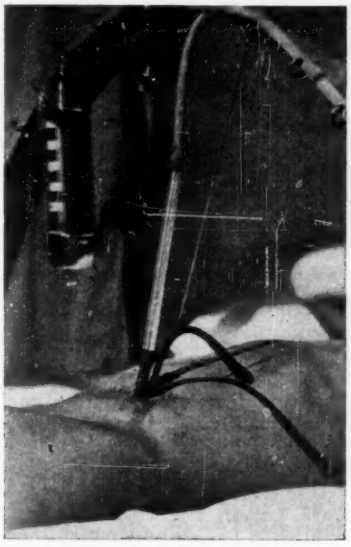


FIG. 7. Photograph of patient shown in figure 5 after increase in weight and diminution of area of excoriated skin about fistula. Outer disc removed, inner disc held in position by traction of rubber thread tied to catheter protruding from inner disc.

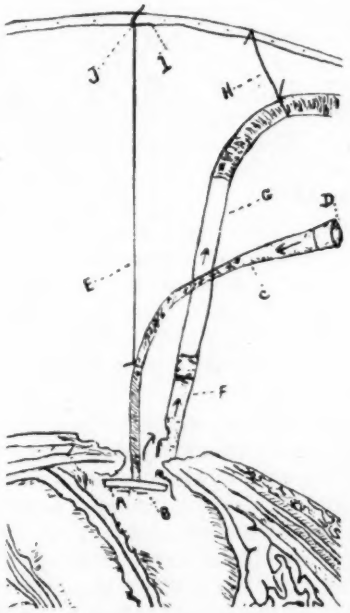


FIG. 8. Diagram of apparatus and cross section of abdominal wall and underlying gut through fistula, illustrating figure 7. A, inner elliptical disc; B, safety-pin; C, catheter; D, cork stopper in end of catheter; E, rubber thread; F, rubber tubing with opening in its side near its lower end; G, section of glass tubing; H, thread supporting suction tube; I, arc of hoop over patient.

ment in the general appearance of the patient was noticeable within forty-eight hours after instituting these measures to check the loss of nourishment. The desire for food gradually returned. The first solid food voluntarily taken was entirely foreign to the pediatrician's idea of a proper dietary, even for healthy children. It consisted of mashed potato fairly well saturated with thick, greasy gravy. The

second article was a small chunk of American cheese. Two or three days later, at the patient's request, a small piece of mince pie was permitted. Strangely enough, these foods appeared to be well tolerated. Forced feeding by the Levine tube was now discontinued twelve days after its inception, and she soon became coöperative in the taking of a rational diet.

Coincident with the improvement of the patient's general condition, there was healing of the partially digested skin and tissues about the enterostomy and appendectomy incisions. With the eroded area of tissues diminished to that covered by the outer disc, the latter was removed to facilitate further healing (figs. 7 and 8). The inner disc was held in place by the upward pull of a somewhat delicate rubber thread made by opening a rubber band. The patient continued to gain weight, and the skin healed to within approximately one-quarter of an inch of the fistulous opening.

Within four weeks after the foregoing measures were instituted, the fistula had become so small and the superficial tissues so well healed that the stoma was blocked by the two-button method and the patient was dismissed from the hospital. After an ambulatory period of eight weeks at home, the patient was again hospitalized, the buttons removed, and a successful extra-peritoneal closure of the fistula made without difficulty. She has remained well two years and ten months, and the abdomen presents neither incisional bulging nor hernia.

Contrary to reasonable expectation, release of the disc did not result from sloughing of tissues about the margin of the stoma. Tension maintained by the rubber thread was just enough to hold the disc in position after the taking of food. For short periods during the day and for longer periods at night, after the stomach had emptied, adjustment of the thread was such that it exerted very little tension. Whether the peristaltic contractions of the bowel sufficiently altered the degree of upward pressure exerted by the elliptical disc on the mucosa to prevent its ischemia and sloughing, or whether the mucosa in contact with the periphery of the disc disappeared, was not determined. Although illogical in principle, it is certain that the position of the disc was maintained by this means for fifteen days.

In terminating my remarks, it may be said that certain innovations introduced in the past two years for the treatment of obstruction of the small bowel have been briefly reviewed, and that fistulous formation incident to the use of enterostomy has been stressed as its most undesirable effect. When such a serious complication arises in the treatment of a surgical condition, it has been suggested that some procedure other than the routine can in certain instances be devised to influence most favorably that which at first appears utterly hopeless.

REFERENCES

1 Cameron, Angus L.: The treatment of duodenal fistula. Surg. Gynec. Obst. 37:599 (Nov.) 1923.
2 Cheever, David: Operative evacuation of the small intestine in paralytic stasis. New Eng. J. Med. 207:1125 (Dec. 22) 1932.
3 Elman, Robert: The treatment of late intestinal obstruction. Surg. Gynec. Obst. 56:175 (Feb.) 1933.
4 Foster, W. C.: Acute intestinal obstruction. J. A. M. A. 91:1523 (Nov. 17) 1928.
5 Orr, Thomas G.: Indications for enterostomy. J. A. M. A. 101:1300 (Oct. 21) 1933.
6 Wangensteen, Owen H.: Therapeutic considerations in the management of acute intestinal obstruction. Arch. Surg. 26:933 (June) 1933.
7 Wangensteen, Owen H., and Paine, John R.: Treatment of acute intestinal obstruction by suction with the duodenal tube. J. A. M. A. 101:1532 (Nov. 11) 1933.

DISCUSSION

DR. EDWARD H. RISLEY, Waterville, Maine: Dr. Webber's paper brings up a most important subject for discussion. From what one reads in the literature of poor results and from what one sees in consultation work, it is rather evident that enterostomy is being rather indiscriminately used in postoperative complications and that an evaluation of its uses is desirable. I will attempt to give these briefly, as I see them.

Contraindications;

1. In general peritonitis with paralyzed intestine, absence of peristalsis defeats the purpose of the enterostomy. It is certainly not indicated as a last resort procedure in general peritonitis as it is so often employed.
2. In adynamic or paralytic ileus, it has no indications, for essentially the same reasons as in general peritonitis. Ileus is best treated by external heat, either dry or moist, by irritative enemata, the judicious use of pituitrin, the use of the indwelling duodenal tube and maintenance of water balance.
3. It is not indicated in high small intestine obstruction, unless the cause of the obstruction is removed at the same time. For an enterostomy above the obstruction will only empty the contents of a small section of high bowel and the loss of chlorides and fluids is so rapid that the patient quickly succumbs in spite of all attempts at replacement.

   Lower in the small intestine, in the region of the terminal ileum, however, the loss of chlorides and fluids is not so rapid and here enterostomy is often of greatest value.

Indications;

Enterostomy is of great value in the acute obstruction complicating acute salpingitis, pelvic abscess and peritonitis. It tides the patient over the acute stage and avoids operation at a most unfavorable time. It is often of great value after relief of obstruction due to strangulation, whether resection has or has not been done. The boggy, edematous, sluggish intestine needs assistance and enterostomy above such an area is often of great service.

These are fairly clean-cut indications for and against the use of enterostomy, and if the surgeon were always careful to make sure that he had a positive indication or contraindication, enterostomy would soon be confined to a certain definite, limited place in the relief of postoperative complications; of extreme value where indicated—as source of definite distress and danger when not indicated.

Dr. Webber has spoken of the value of the indwelling duodenal tube. I should like to emphasize its value still further for it seems to me that here is a little instrument of greatest value; the indications for the use of which are constantly widening.

Looking back over past years I wonder how we ever tided over many of our postoperative compli-

cations without it. In the first place it gives such immediate and generally permanent relief to the patient and relieves the surgeon of the arduous labor of having to pass the old-fashioned stomach tube at frequent intervals. These two advantages alone make it worth while.

Indications;

1. In practically all cases of prolonged nausea and vomiting from whatever cause.
2. In all cases of suspected acute postoperative dilatation of the stomach.
3. In adynamic ileus and especially in cases of suspected postoperative obstruction. It is a well-recognized fact that obstructions may be segmental at first, as when a loop of intestine lies adjacent to an inflammatory area, and that if this intestine can be decompressed before further congestion and paralysis take place, complete obstruction may be aborted and the patient saved from the often fatal secondary operation for complete obstruction. In such cases the early use of the duodenal tube and the administration of adequate amounts of fluids and chlorides are life-saving procedures. It is in such cases that the duodenal tube has its greatest value.

   Lately we have found several cases with postoperative nausea and vomiting and hiccough, in which this latter troublesome symptom was immediately relieved by the duodenal tube.

DR. ALBERT A. BARROWS, Providence, R. I.: On hearing Dr. Webber's very interesting, carefully prepared paper, and reviewing the recent literature on the values and also some of the disadvantages of enterostomy, certain things stand out quite distinctly.

No one will question the fact that in certain cases enterostomy may be a life-saving procedure. Few will question the value of temporary drainage by means of a Levine tube. No one, probably, believes that an enterostomy is of any value in cases of general peritonitis, and no one will deny that such a procedure may be of inestimable value as a preliminary to operation for obstructing tumors of the large bowel, and other intestinal obstructions, even severely toxic cases mechanically obstructed.

These observations have been interestingly borne out by a review of about forty cases in which an enterostomy was done at our hospital between the years of 1929 and 1933.

I feel sure that I must have missed a good many cases of enterostomy that were done. However, after a review of these cases and a tabulation of our results, I was immediately impressed with the striking similarity between the results that we obtained and the results which led to the observations I have just read.

Of this series approximately one-third were cases of paralytic ileus and general peritonitis, in which the enterostomy was done as a last resort, cases which Dr. Risley has called to your attention, in which it is of no value whatsoever. All these cases died.

The second group of cases was a little group of six in which enterostomies were done for acute obstructions, in which there was either no peritonitis or in which the peritonitis was of definitely local character. Of these six cases, four recovered, a fifth had apparently recovered from his obstruction but died from an acute cardiac condition at about the tenth day. The sixth case died from shock, from resection of about seven feet of bowel, the operator apparently feeling at the time that the resection was necessary.

The remaining cases of this series consisted of obstructions, partial or complete, from tumors, usually carcinoma of the large bowel, in which an enterostomy was done. Recovery took place in a little less than half of these cases, and in this series I had to include one case in which the tube was accidentally withdrawn by the patient and a peritonitis followed, and death.

I believe that enterostomy will continue to have a very valuable place in emergencies and should not be overlooked, and that it may act to prevent the surgeon from attempting too much at an inopportune time.

LEUKOPLAKIA BUCCALIS AND CANCER*

BY SOMERS H. STURGIS, M.D.,† AND CHARLES C. LUND, M.D.†

INTRODUCTION

THE end results in 248 selected cases of leukoplakia and keratosis of the mouth or lip entering the Collis P. Huntington Memorial Hospital during the years 1918-1926 have been previously reported[1]. These cases showed no malignant disease upon entry, and were observed for a period of five years or more. It was calculated from Massachusetts Death Rates for males from 1920-1930, that, in such a group, but one death from buccal cancer might be expected on the basis of chance alone. There were actually, however, twelve deaths among the twenty-five patients of the series who developed buccal cancer after entry. Considering the statistical method that was used, it is reasonable to conclude that cancer developed at least twelve times more frequently in association with these lesions than might have been expected in any similar group of normal individuals without such lesions.

The present communication is a more detailed study of the twenty-five patients that developed malignancy of the mouth or lip while under treatment for precancerous lesions of the same areas. Since these cases were often under observation for a considerable time before the appearance of malignant disease, it was thought that such a study might throw light on the importance of the factors generally considered etiologically significant in the development of buccal carcinoma. These factors have been stud-

*From the Cancer Commission of Harvard University. This work was made possible by grants from anonymous donors and from the Delamar Mobile Research Fund.

†Sturgis, Somers H.—Member of Staff, Out-Patient Department, Cambridge Hospital. Lund, Charles C.—Surgeon, Collis P. Huntington Memorial Hospital. For records and addresses of authors see "This Week's Issue," page 32.

ied by contrasting their occurrence in the twenty-five cases progressing to cancer, with their occurrence in the 223 others that did not develop malignancy.

Twelve of the twenty-five cancer patients died; this group represents the failures in the treatment of the 248 cases of premalignant disease. The records of these twenty-five cases have been analyzed to emphasize the significant cause or causes in each for the fatal result compared with the good result.

SIGNIFICANCE OF ETIOLOGICAL FACTORS

The factors that are widely recognized as of etiological importance in the cause of both premalignant disease and cancer of the mouth or lip are age, sex, syphilis, the use of tobacco, and poor dental hygiene. The following table shows that no significant differences appear when the occurrence of these factors among the cases that developed cancer is compared with their occurrence in the rest of the cases.

TABLE 1

OCCURRENCE OF ETIOLOGICAL FACTORS IN 248 CASES OF PREMALIGNANCY

Factors	No Cancer— 223 Cases	Cancer— 25 Cases
Age	54 years	60 years
Sex—Males	91%	96%
Syphilis	6%	4%
Use of Tobacco	94%	85%
Bad Dental Hygiene	92%	80%

These etiological factors, therefore, are of no value in predicting whether a case of premalignant disease is likely to develop cancer.

SIGNIFICANCE OF LOCATION, DURATION AND EXTENT OF PREMALIGNANT DISEASE TO CANCER DEVELOPMENT

Location. In the present series there were fifty-three cases of leukoplakia of the inside of the mouth, including lesions on the tongue, the palate, or the floor, and inside the cheeks. The remaining 195 patients showed leukoplakia or keratosis involving the lips only. In table 2 the incidence of cancer in leukoplakia inside the mouth is seen to be twice as great as that in premalignant lesions of the lips only.

TABLE 2

LOCATION OF PREMALIGNANCY AND CANCER INCIDENCE—248 CASES

Location	Total Cases	Cancer Cases
Lips only	195	15 — 8%
Inside the Mouth	53	10 — 19%

This tends to confirm the clinical impression that leukoplakias inside the mouth respond less readily to treatment and are more apt to show obstinate persistence than similar lesions confined to the lips.

Duration. Duration of the premalignant lesion before treatment in the clinic, does not seem to have any relation in this study to the development of malignancy. When the whole series of cases is divided into groups according to duration of their disease before entry to this hospital, no correlation can be found between the more chronic groups and the incidence of cancer[1]. The twenty-five patients that developed cancer averaged one year and seven months from onset of premalignant disease to first treatment in the clinic; the rest averaged one year and eleven months for the same interval. The cancer patients as a group, then, showed no greater neglect of their premalignant condition before applying for treatment.

Extent. In 216 of the 248 cases it was possible to classify the premalignant lesions from the data in the records relating to their size or extent. Multiple, diffusely scattered leukoplakias involving individually but small areas of buccal mucosa, are included in the "large" or more extensive group of cases. Table 3 shows the incidence of cancer in relation to size of leukoplakia or keratosis.

TABLE 3

EXTENT OF PREMALIGNANCY AND CANCER INCIDENCE—216 CASES

Size of Lesion	Total Cases	Cancer Cases
Small: 0.1-0.5 cm.	65	1 — 2%
Medium: 0.6-2 cm.	80	8 — 10%
Large: 2.1 cm.	71	16 — 22%

An appreciable increase in cancer incidence is found in premalignant lesions of greatest extent at the time of the first clinic examination.

FACTORS OF IMPORTANCE IN THE END-RESULTS OF THE CANCER CASES

Theoretically, of any group of cases treated by a cancer hospital, those that are seen before cancer begins, and observed constantly during the treatment of the premalignant lesion, should give the best results of treatment when actual cancer develops and is treated. That as many as twelve out of twenty-five such cases should prove fatal has prompted us to analyze the factors of possible importance leading to these failures. Of the twenty-five cases in which cancer developed, fifteen were located on the lip and ten involved the mucous membrane of the mouth. There were six fatalities in each group. Forty per cent of failure in cases of carcinoma of the lip and 60 per cent of failure with carcinoma of the mouth are results no better than those obtained in other series of cases that did not come under observation during the premalignant phase of their

disease. The various factors leading to these unfavorable results follow:

1. Delay. Many patients with persistent or suspicious premalignancy will neglect routine follow-up visits even in the face of the most constant urging. Delay was considered important when the diagnosis of cancer was first made after an interval of more than three months from the preceding visit. With this criterion it was found that in eight out of the twelve fatal cases, there was delay in the early discovery of malignancy. In comparison, seven out of the thirteen cases that were "cured" had also neglected their routine visits.

2. Extent of premalignancy. Since cancer developed more frequently in the cases with widespread leukoplakia, the records were studied to find the influence of this factor on the fatal outcome. There was an extensive premalignant lesion in eight out of the twelve fatal cases, and in seven out of the thirteen non-fatal cases.

3. Metastases. In five fatal, and one "cured" case, metastases were already present upon the first discovery of malignancy. Delay in reporting to the clinic was also a factor in all but one of these six cases.

4. Malignancy of Cancer. Twenty of the cancers in the group of twenty-five cases under study were of low-grade malignancy. Strangely enough, only one of the five more malignant tumors resulted fatally.

5. Inadequate Treatment. Of the group of twelve fatal cases there were three patients so feeble at the time carcinoma was discovered that the optimum treatment could not be given without too great a risk. It is also apparent from the records that three other cases in this group received definitely inadequate treatment.

6. Multiple Malignancy. In one fatal case and one "cured" case there were multiple foci of primary cancer.

7. Error in Diagnosis. In one fatal and one non-fatal case, there is evidence that the diagnosis of cancer was not made at the earliest possible visit to the clinic.

SUMMARY OF IMPORTANT FACTORS

The factors discussed above may be classified in two groups; those that are primarily dependent on the human equation and therefore alterable, and those that are beyond control in the present state of our knowledge. The first group includes the element of delay on the patient's part, as well as the failure of the physician to diagnose or to treat adequately the cancer at the earliest possible visit. The second group of factors includes early metastasis, extent of premalignant lesion, high grade of malignancy of tumor, and multiple foci of primary carcinoma. In this small series, the factors classified in the first group appear to have been more important in determining the outcome than those in the second. Thus, four patients in the group of twelve who later died of their disease, having neglected routine visits, returned only after metastases were already present. Two others who delayed their return, whose feeble condition precluded radical treatment, came back too late for a minor procedure to offer any hope for cure. In four cases inadequate treatment was apparently given, and in one the chance to make an early diagnosis was missed. The fatal outcome in each case might have been prevented by the elimination of such factors as these. On the other hand, the malignancy of the cancer in this group of patients, unlike that in other larger series of carcinomas, was not important in the end result. The presence of metastases when first seen was generally the result of delay. There were about as many extensive leukoplakias among the patients that were cured as among those that died. The factor of multiple malignancy was not found to be significant in these cases.

In so far as any conclusions may be drawn from so few cases, it is felt that the disappointing results here obtained can be improved upon, since the fatalities were to a certain extent dependent on preventable causes.

CONCLUSIONS

1. The occurrence of the etiological factors usually accepted as important in the development of premalignant lesions of the mouth or lip is of no value in predicting the likelihood of cancer formation.

2. The cancer incidence in leukoplakia of the mucosa inside the mouth is definitely higher than that in premalignant lesions confined to the lips.

3. The incidence of oral cancer is not increased in the premalignant lesions of longest duration.

4. In the group of cases under study, there is a definite correlation between cancer incidence and extent of the premalignant disease.

5. From a study of the factors involved in the fatal cases, it is seen that in all but one of the twelve fatal cases there were one or more "preventable" factors leading to the poor result. In order of importance these were, delay in reporting, inadequate treatment, and error in diagnosis.

6. In other words it is not the syphilis, tobacco, poor teeth, etc., that make a case hopeless but the stage at which it reaches the physician and the treatment given immediately when first seen.

REFERENCE

1 Sturgis, S. H., and Lund, C. C.: Leukoplakia buccalis and keratosis labialis. New Eng. J. Med. 210:996 (May 10) 1934.

ACUTE ANAPHYLACTIC SHOCK FOLLOWING INTRACUTANEOUS TEST FOR SENSITIVITY TO HORSE SERUM*

Report of a Fatal Case

BY HAROLD J. FREEDMAN, M.D.†

THE literature reveals a moderate number of reports of sudden death following the injection of a foreign protein into a hypersensitive individual. Lamson[1] reported forty-one fatal cases; eight of the patients had received a previous injection of horse serum. Lord[2] reported nine fatal cases. Gottstein[3] reported twelve cases and Gillette[4] reported sixteen. Fatal reactions following the subcutaneous, intramuscular or intravenous injection of horse serum comprise the largest group of such anaphylactic deaths. Fatal reactions following the intracutaneous injection of a protein, however, are extremely rare. Baagoe[5] described a fatal reaction in a nine year old child who had received 0.1 cc. of egg yolk as a skin test. Dyspnea, collapse and death occurred without the appearance of urticaria. Lamson[1] reported a five months old eczematous infant sensitive to egg who became cyanosed and edematous and died, without the appearance of urticaria, two hours after a scratch test with 0.05 cc. of ovocoid. Cooke[6] reported a three year old asthmatic boy who became cyanosed and died, without the appearance of urticaria, a few minutes after a skin test with 0.02 cc. of glue (0.0001 gram of nitrogen per cc.). Lamson[7] reported a thirty-four year old asthmatic male who died without urticaria five minutes after being tested with buckwheat.

The following experience which occurred after an intradermal test for sensitization to horse serum seems worthy of report:

The patient was a six year old boy who for a period of years had suffered from recurrent attacks of *asthma* and *eczema*. Twenty days before coming to the clinic he received an injection of diphtheria toxin-antitoxin at school. Six days later he suffered a severe asthmatic attack lasting three days. The mother thought that the asthmatic attack and subsequent urticaria were directly the result of the injection of toxin-antitoxin. The asthmatic attack was followed in three days by a severe urticarial reaction. In order to test the patient for sensitivity to horse serum, an intracutaneous injection of 0.05 cc. of horse serum was given. Two minutes after the material was introduced into the skin a large wheal formed at the site of injection and urticaria appeared on the face and body. Five minims (0.3 cc.) of adrenalin chloride were injected into the left arm. Approximately one minute later the child doubled up and complained of abdominal pain. In a few seconds he became intensely cyanosed and respirations ceased. Emergency treatment consisted of further injections of adrenalin chloride both subcutaneously and into the heart, manual artificial respiration and the use of the Drinker respirator. These measures were ineffective. The child died about eight minutes after the test was done; he had received over 1 cc. of adrenalin chloride before respiration ceased. Necropsy examination was not obtained.

SUMMARY: A fatal case of anaphylactic shock following the intracutaneous injection of horse serum is reported.

*From the Department of Pediatrics, Harvard Medical School, and the Infants' and Children's Hospitals, Boston, Mass.

†Freedman, Harold J.—Assistant Physician, Medical Department, Children's Hospital. For record and address of author see "This Week's Issue," page 32.

REFERENCES

1 Lamson, R. W.: Sudden death associated with the injection of foreign substances. J. A. M. A. 82:1091 (April 5) 1924.

2 Lord, F. T.: Serum disease and serum accidents. Am. J. Ophth. 11:451, 1928.

3 Gottstein, A. J.: Ueber Todesfälle, welche bei der Anwendung des Diphtherieheilserums beobachtet worden sind. Therap. Monatsh. 10:269, 1896.

4 Gillette, H. F.: Diphtheria antitoxin in bronchial asthma. J. A. M. A. 50:40 (Jan. 4) 1908.

5 Baagoe, K. H.: Case of allergic death due to intracutaneous injection of 10 c.c. of white of hen's egg. Ugesk. f. Laeger. 90:550, 1928.

6 Cooke, R. A.: Studies in specific hypersensitiveness. III. On constitutional reactions: the dangers of the diagnostic cutaneous test and therapeutic injection of allergens. J. Immunol. 7:119, 1922.

7 Lamson, R. W.: So-called fatal anaphylaxis in man. J. A. M. A. 93:1775 (Dec. 7) 1929.

THE RELATION OF ARCUS SENILIS TO ARTERIOSCLEROSIS AND SENILITY

BY CHARLES E. WHITE, M.D.*

FOR many years the well-known condition called arcus senilis has been written about in textbooks and journals. The name given to this condition, i.e., arcus senilis, has been accepted with little discussion. The name would indicate that this condition is related to senility. Most every physician has seen many senile cases without it, and probably has seen young people with it. This observation alone would indicate that the term "arcus senilis" is misleading if the name itself is to denote the character of this condition.

In 1927, V. C. Kinney offered clinical evidence of the non-relationship of arcus senilis to the so-called diseases of old age; however, clinical evidence is frequently contradicted or altered by postmortem findings. On the contrary, E. Stromgren offered evidence that arcus senilis occurred in the pyknic constitutional type with a frequency that would tend to show a definite

*White, Charles E.—For record and address of author see "This Week's Issue," page 32.

relationship between the two. In a roundabout way, Stromgren links up the pyknic constitutional type, hypercholesterinemia, arteriosclerosis and arcus senilis, accepting arcus senilis as related to arteriosclerosis. Some present-day psychiatrists mention it as being a sign of cerebral arteriosclerosis. To be a sign significant of any condition, a sign must occur in at least a liberal majority of cases showing that condition. Conversely, if cases not showing the sign have the condition for which the sign is considered significant, then the significance of the sign is lessened or destroyed.

The writer, wishing to determine whether arcus senilis bears a definite relation to a number of pathological conditions, has taken four hundred cases on which physical examinations, mental examinations, laboratory work, and finally postmortem examinations have been done, and correlated the findings. The postmortem examinations include examination of the brain both microscopically and grossly. The four hundred cases include one hundred and thirty-four cases with arcus senilis and two hundred and sixty-six cases without it. The ages of the group range from twenty-three to ninety-two years inclusive. The cases all suffered from mental aberrations or diseases, and are divided among two hundred and forty-one males and one hundred and fifty-nine females. The findings are worked out on a percentage basis.

The pathological conditions considered having a possible relation to arcus senilis:—

1. Clinical Arteriosclerosis
2. General ”
3. Cerebral ”
4. Nephritis
5. Hypertension

Clinical Arteriosclerosis: Any demonstrable sclerosis, tortuosity or beading of the radial, brachial, temporal or dorsalis pedis arteries.

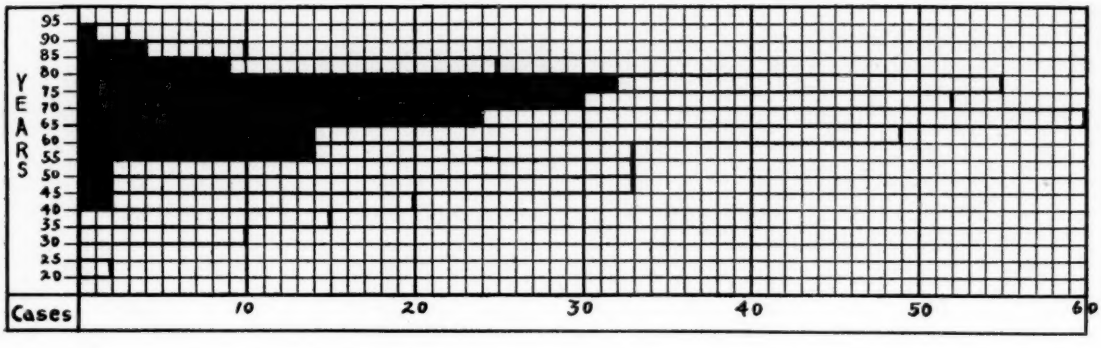
General Arteriosclerosis: Definite sclerosis of coronaries or of the aorta and its larger branches or both.

Cerebral Arteriosclerosis: Definite sclerosis of basal arteries of the brain, of the Circle of Willis or its branches, or microscopic occluding sclerosis of the smaller subdivisions.

Nephritis: Any case showing the urines of nephritis or postmortem nephritic kidneys.

Hypertension: Any case showing above 150 mm. of mercury.

CHART 1.



Total No. Cases.

No. Cases with Arcus Senilis.

“ “ without “ “

	3rd	4th	5th	6th	7th	8th	9th	10th
Percentage of Arcus Senilis in each Decade—	0.0	0.0	7.6	24.2	34.9	57.9	37.1	33.3

“ “ “ “ “ 6th “ and Upward 40.6
“ “ “ “ “ 8th “ “ “ 52.4
“ “ “ “ “ 5th “ “ Below 5.0

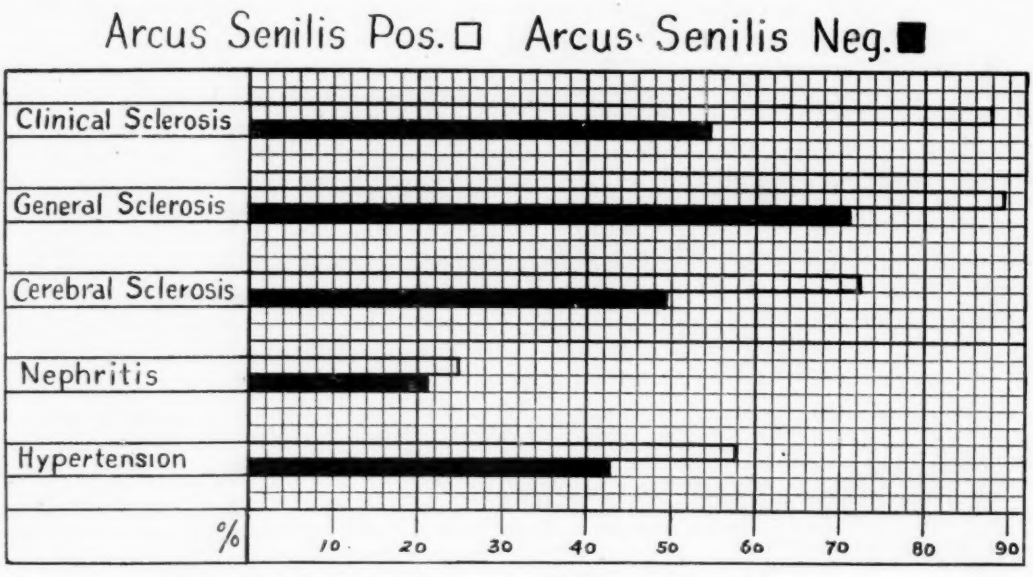
Of the four hundred cases, one hundred and thirty-four showed a positive arcus senilis or 33.5 per cent. Considering the sixth decade as a period when arteriosclerosis begins to lay down demonstrable pathology, 80 per cent of the cases died during or after this decade. Chart 1 shows the age distribution of the four hundred cases as well as the age distribution of the cases showing arcus senilis.

It is obvious that as 59.4 per cent of the cases in the sixth decade and beyond show no arcus senilis, the 40.6 per cent of the cases that do show arcus senilis must be accounted for on a basis other than that of arteriosclerosis.

Similarly with 47.6 per cent of the cases in the eighth decade and beyond showing no arcus senilis, the 52.4 per cent that do show it must be accounted for on a basis other than that of senility. This being true the 5 per cent of cases below the sixth decade showing arcus senilis must be accounted for on a basis other than arteriosclerosis or senility.

The pathological conditions being accounted

CHART 2.

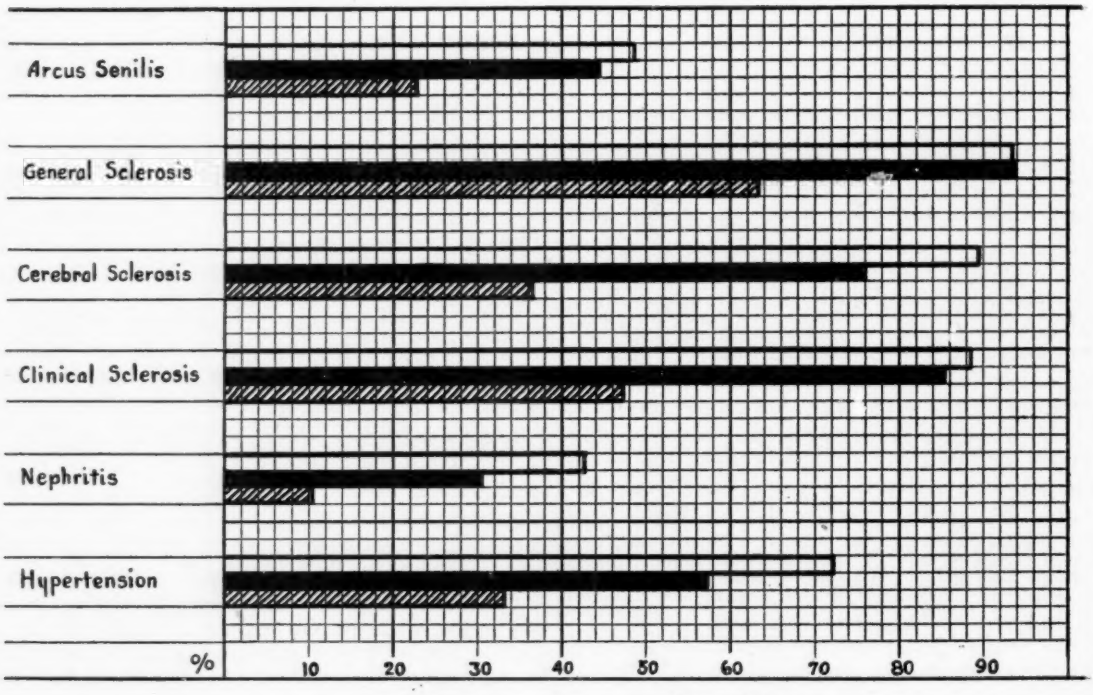


The positive Arcus Senilis starts at 20 years higher than the negative cases.

Positive Arcus Senilis margin of	Clinical Sclerosis	33.5%
	General "	18.4%
	Cerebral "	22.7%
	Nephritis	3.2%
	Hypertension	15.7%

CHART 3.

Psychosis *c* Cerebral Arteriosclerosis □ Senile Psychoses ■
Other Psychoses ■



Psychoses	Age at Death		
	Youngest	Oldest	Avrg.
Psychosis *c* Cerebral Arteriosclerosis	40	88	69.5
Senile Psychoses	55	91	76.3
Other Psychoses	23	92	56.3

for are also found in cases showing no arcus senilis. In chart 2 a comparison of percentage of occurrence of these conditions is shown in cases with and without arcus senilis.

It must be remembered that in this group the cases with arcus senilis start about twenty years higher than the cases without arcus senilis; therefore, the pathological conditions considered must necessarily show a high incidence in the positive cases because of the age difference. Even then the margin is not great enough to be considered significant.

All of the cases in this group were cases with mental disorder, so that a comparison of the percentage of occurrence of the several pathological conditions in related psychoses is of interest. It will be seen in chart 3 that arcus senilis was present in but 48.5 per cent of the cases diagnosed psychosis with cerebral arteriosclerosis, and in but 44.5 per cent of the cases diagnosed as senile psychoses. In these two psychoses the pathological conditions that appeared to be related from their frequency of occurrence were general arteriosclerosis, cerebral arteriosclerosis, clinical arteriosclerosis, and in lesser degree, hypertension.

Arcus senilis was found to be present in:

Clinical	Arteriosclerosis	44.7%
General	"	38.8%
Cerebral	"	42.4%
Nephritis		36.7%
Hypertension		40.8%

With arcus senilis occurring in less than 45 per cent of the cases showing any of these pathological conditions, it cannot be considered as a sign of any one of them.

CONCLUSIONS

1. The term "arcus senilis" does not denote the condition for which it is named, and does not occur in cases having reached the senile age in high enough percentage to be considered a sign of senility.
2. Arcus senilis cannot be considered a sign of general arteriosclerosis, cerebral arteriosclerosis, nephritis, or hypertension because of the low percentage of its occurrence in cases showing these pathological conditions.

REFERENCES

1 Kinney, Virgil C.: Arcus senilis. Studies pertaining to the depigmented circles of the iris and their non-relationship to the so-called old age diseases. Internat. J. of M. & S. 40: 152 (April) 1927.
2 Stromgren, E.: Constitutional type and arcus senilis. Acta psychiat. et neurol. 7: 823, 1932.

MEDICAL PROGRESS

PROGRESS IN NEUROLOGY IN 1933

BY JULIUS LOMAN, M.D.*

MANY excellent neurological studies not only on clinical but also on purely anatomicophysiological problems have been reported during the year 1933. The baffling problems presented by epilepsy and migraine continue to receive great physiological consideration, productive on the whole of clearer understanding. More and more attention is being given to therapeutics with interesting results, particularly in the muscular dystrophies and myasthenia gravis, in migraine, in chorea minor, in disturbances of the sympathetic nervous system, and in some functional nervous conditions.

Anatomicophysiological Studies

Action Currents in the Central Nervous System

For the past year Saul and Davis[1] have been working on the effect of action currents on the central nervous system. As they state, the central nervous system may be thought of as an organization of tracts and centres which transmit and integrate nerve impulses. Nervous and mental diseases may be considered as disorders of such transmission and interplay. Thus, a method for detecting and studying the nerve impulses in the functioning nerve tissue might throw some light on the workings of the nervous system in health and disease. The authors explain that nerve impulses are invariably associated with electrical disturbances, called action currents. One is never found without the other; they are interchangeable but not proved to be identical. The action current travels at a speed of fifty to one hundred meters a second and derives its energy from the nerve itself.

The technique utilized by the authors is as follows: A stimulus is applied to a sense organ, as a light to an eye. This results in a localized flow of nerve impulses along the appropriate tracts of the central nervous system. Electrodes placed on the proper tracts (for example, the optic tract) detect the action currents. The disturbance thus detected is amplified by electron tubes and recorded on a special recording device. These action currents may then be seen and heard and permanently recorded for later study. The brains are sectioned for anatomic correlation.

The following results were obtained: The observations indicate two distinct electrical phenomena involved in hearing: true action currents and "spread". This spread is an electric

*Loman, Julius—Instructor in Neurology, Tufts College Medical School. For record and address of author see "This Week's Issue," page 32.

current which seems to originate in the cochlea, which is conducted throughout the tissues of the head at the usual speed of electricity. The action currents are highly localized in the auditory pathways. Musical tones applied to the cat's ear are reproduced in the loud speaker only up to 1000 double vibrations per second; words come back blurred. Again, electrodes may be placed on the optic tract. This preparation is so sensitive that in a diffuse light a foot from the cat's eye, the amount of movement necessary to pinch a small forceps stimulates the retina to produce action currents enough in the optic tract to give a definite response on the oscillograph and loud speaker. Olfactory experiments were not conclusive.

Cerebral Cortical Localization

Adrian[2] makes some very interesting remarks on the activity of nerve cells. He stresses that it is upon the total mass of functionally active cortex and not upon its distribution that learning depends. Conversely, the effects of an injury in terms of loss of responses depend upon the amount of gray matter destroyed and not upon the interruption of specific cortical connections. Thus, the central nervous system of a mammalian vertebrate is capable of relearning acquired habits that have been lost through experimental destruction of those parts from which these habits are normally localized. These acquired responses of the cortex can nearly all be learned, and nearly all destroyed responses can be relearned in any part of the original or of the residual cortex, respectively.

The Dermatomes in Man

As a result of observations during his long surgical experience, Foerster[3] has been able to define all the dermatomes of the lower extremity, that is, to make an accurate determination of the distribution of the posterior spinal roots in man. To define a dermatome, Foerster utilized two methods, one of which he calls the constructive method. This consists of dividing a series of contiguous roots. The superior border of the resulting anesthesia represents the inferior border of the dermatome, which corresponds to the next higher intact root, whereas the inferior border of the anesthetic area represents the superior border of the next lower dermatome. The second method used by Foerster, first utilized by Strecher and Bayliss on animals, is based on the fact that faradic stimulation of the distal part of a divided posterior root is followed by vasodilation. Foerster reports the following results of his observations:

1. The dermatomes of man overlap to the same degree as do those of the monkey. Foerster has never found that resection of one single root in man was followed by loss of sensibility, as detected by the usual clinical methods.

2. The tactile dermatomes are larger than the pain and thermal dermatomes. Sometimes the resection of two contiguous roots produces analgesia and thermanesthesia but no tactile anesthesia.

3. The areas of vasodilation produced by electrical stimulation of the posterior roots are similar but not identical with them. The former are similar to the areas of herpetic eruption.

4. A single dermatome is represented not only in the totality of the corresponding root but in every single filament of the root. When all filaments, except one of each of a number of roots are cut, no anesthesia results; each of the corresponding dermatomes preserves its sensibility.

Epilepsy

Etiology

Probably the most significant study on the pathological physiology of epilepsy this past year is that of Penfield[4]. This author observed the brains of thirty patients during convulsions, in whom electrical stimulation in the brain was made on the operating table. Twenty-six of these patients showed striking vascular changes and all but six showed some alteration in cerebral blood vessels or in vascularization as sequel to the attacks. One constant visible phenomenon in the brain during an epileptic seizure, according to Penfield's observations, is cessation of arterial pulsation. Cerebral pallor may be present during the seizure but more often follows it. The epileptic brain is subject to local vasomotor reflexes, such as have never been described in the normal brain. Physiologic instability of the blood vessels seems to be the abnormal state common to epileptics of all varieties.

The vasomotor spasms and changes seen so characteristically in the cerebral cortex of epileptics are due, Penfield states, to vasomotor reflexes, but reflexes which are probably not subserved by autonomic centers placed outside the cranial cavity; for cervical and dorsal sympathectomy should make epilepsy impossible if this were so. This operation, performed by Penfield on four patients, did not abolish seizures. The vascular abnormality, Penfield finally concludes, is thus probably centered in the brain itself.

Lennox[5] emphasizes that the "all or none" conception of etiology is no more true for epilepsy than for other medical conditions. Lennox stresses that the physician must seek not simply a cause but the various causes of seizures. He likens epilepsy to a reservoir of waters, the deepest portion of which is occupied by the patient's susceptibility, a tendency presumably inborn or constant. With this as a basis, there is a periodic overflow of the restraining banks by feeding springs. These springs represent the contributory factors of

epilepsy and include brain lesions, emotional disturbances, sympathetic and somatic abnormalities. By discovering the incoming springs and raising the banks, that is to say, increasing the seizure threshold, the waters are kept below the overflow level.

Treatment of Epilepsy

Disagreement regarding the efficacy of restriction of fluid intake in the treatment of epilepsy continues. Fremont-Smith and Merritt[6] agree with the negative side taken by Stanley Cobb. The former authors' experiments show that well-marked variations in the level of fluid intake in man have no appreciable effect on the cerebrospinal fluid pressure; and further, that the influence of the level of the fluid intake upon the incidence of convulsive seizures is not dependent upon the variations in intracranial pressure.

Fay, however, is still convinced that dehydration, a theory presented five years ago, actually produces a decrease in cerebrospinal fluid pressure, when properly maintained, and that regulation of fluid intake can be accomplished only when all the factors, including the diet, are carefully controlled.

Lyon and Dunlop[7] have observed the therapeutic effects of dehydration upon a small series of epileptic patients having seizures every day. During the periods of dehydration, definite beneficial effects were noted in four out of five cases. This improvement compared favorably with that produced by treatment with ketogenic diet, but was not so good as that effected by adequate doses of luminal. The effects of luminal, however, were enhanced by maintaining a state of dehydration.

On the other hand, Fetterman and Kumin[8] treated twenty-one epileptics by dehydration with intakes as low as 100 cc., and yet their results were essentially negative. The change to hydration did not with any regularity precipitate attacks.

In commenting on the results obtained by the above authors, Fay remarks that it is dangerous to reduce a patient's fluid to 100 cc. a day. The objective in the dehydration treatment is not to subtract fluid to an intolerable degree, Fay states, but to subtract fluid from the patient's body fluid reservoirs. Unless the patient demonstrates an actual loss of body weight, there is no subtraction or real dehydration. Fay outlines a regimen for accomplishing this aim.

Treatment of Status Epilepticus

Storchheim[9] obtained very good results in eight cases of status by the use of magnesium sulphate given intravenously. Ten cc. of a twenty-five per cent solution, a safe dose, was found to terminate most attacks of status epilepticus. If necessary, this dose may be repeated and possibly a third time in well-developed individuals. Pulmonary edema, occurring in status, is apparently successfully coped with by the same method of therapy.

Migraine

Etiology

Riley, Brickner, and Kurzrok[10] make a very interesting preliminary report on their hormonal study of migraine. Examination of the urine of eleven female patients during the migrainous attacks showed that the female sex hormone was usually absent, or present in much reduced quantity. Among these patients, a total of twenty-nine headaches occurred during the period of observation. Twenty of the headaches were preceded in their onset by the appearance of prolan in the urine. Prolan also appeared in the urine of two male patients. Injection of follutein caused the appearance of the characteristic attack of migraine in seven out of nine female cases. The authors, therefore, believe that the presence of prolan in the urine is definitely related to the occurrence of migrainous seizures. They further conclude that these results substantiate the hypothesis that ovarian, and presumably hypophyseal, activities are closely related to the occurrence of migraine.

A review of migraine is made by Critchley and Ferguson[11]. These authors distinguish bilious, ocular, menstrual, cerebral, and allergic forms, although they state the evidence for the last type is insufficient. They are cautious about the relationship between migraine and epilepsy. From a therapeutic point of view they find it convenient to distinguish five principal and a few minor types (1) alimentary, including biliary, "duodenal", and abdominal migraine; (2) dietetic, closely related to the former; (3) metabolic associated, as the case may be, with acidosis, alkalosis, or hypoglycemia; (4) allergic; (5) vasomotor and sympathetic, with calcium for the former and ergotamine tartrate for the latter as useful remedies. Among other "types" are those associated with ocular defect, sinus disease, and a "para-epileptic" type. As a final therapeutic suggestion, after mention of the usual remedies, they make the following startling statement: "For the very severe cases which do not respond to the above measures we would recommend a right subtemporal decompression. Gordon Holmes says that he has never known migraine to persist in a patient who has had a surgical or traumatic decompression."

According to Hahn[12], the following succession of events occur in migraine: (1) angiospasm with constriction of the vessels, (2) secondary vasodilation due to the action of locally formed substances; (3) exudation into the tissues as a result of vasodilation. Hahn admits

that frequent occurrence of angiospasm predisposes in later life to organic disease of the vessels concerned; for example, he suggests that there may be the same relation between simple migraine and later hemiplegia as between angina pectoris vasomotoria and coronary thrombosis.

Treatment of Migraine

While working on the action of chondroitin sulphuric acid in peptic ulcer, Crandall and Roberts[13] incidentally learned that some of the patients were relieved of their habitual headaches. Later giving this substance to forty-two patients having periodic headaches, they found that more than half were greatly benefited. The amount of chondroitin sulphuric acid usually given was 3 Gm. daily, either in one dose or in divided doses, in powder or in capsules. The chief constituent of chondroitin is glycuronic acid, which is known to act favorably in cases of hepatic insufficiency; and some investigators have stressed the part of disordered liver function in migraine.

Kottmann[14], who believes that migraine is most frequently caused by localized increased sympathicotonus of vessels, recommends ergotamine tartrate, which is the active alkaloid of secale cornutum isolated by Stoll in 1918. This substance has a strong inhibiting action on the sympathetic nervous system and has been used for migraine since first recommended by Maier of Zürich in 1926. It may be given in doses of 0.5 cc. hypodermically at the beginning or even at the height of the attack. It is also effective in stopping attacks, if given in tablet form by mouth; three tablets a day for a long period have prevented attacks. It is worthless in cases on a vagotonic basis.

Cerebrospinal Fluid

The Rôle of Ventricular Dilation in the Spread of Infections from the Subarachnoid Space into the Ventricles

Flexner[15] states that it is a generally accepted view that there is a steady flow of the cerebrospinal fluid from the ventricles into the subarachnoid space. Probably in part because of this view tubercles of the ventricular ependyma have been thought to occur only when bacilli pass from the blood through the choroid plexus into the ventricles. There is, however, abundant evidence from the studies of Rich and McCordock, who demonstrated that tuberculosis of the ventricles results in the absence of the blood stream infection, that such infection must be accounted for by a retrograde passage of bacilli from the subarachnoid space. In further support of this view Flexner, who points out that infections of the ventricles have been observed to follow primary infections of the meninges, made measurements in the cat of the volumes of the ventricles and the subarachnoid space and of the quantity of the cerebrospinal fluid which may be aspirated from subarachnoid space into the ventricles by ventricular dilation. A comparison of these volumes indicates that a significant volume of fluid may be sucked into the ventricles, and offers an explanation for the retrograde passage of bacteria and other foreign matter from the subarachnoid space into the ventricles.

Relationship of Arterial Blood Pressure to Cerebrospinal Fluid Pressure

Comparing the arterial blood pressure and the cerebrospinal fluid pressure in one thousand four hundred and eighteen cases which were observed at the Boston City Hospital, Fremont-Smith and Merritt[16] conclude that in uncomplicated cases, there is no relationship between the cerebrospinal fluid pressure and the arterial blood pressure, whether systolic or diastolic. There was found to be a definite increase in cerebrospinal fluid pressure in cases of uremia and congestive heart failure. An increase in the intracranial pressure has no effect on the arterial pressure, until the level of the cerebrospinal fluid pressure exceeds that of diastolic pressure. There were six uncomplicated cases of arterial hypertension, in which the cerebrospinal fluid pressure was over 200 mm. The authors have no explanation for this.

Use of Sodium Amytal for Prevention of Lumbar Puncture Headache

According to the experience of Kulchar and King[17], the use of sodium amytal, 3 gr. one-half hour before the puncture, reduces the incidence of reactions after lumbar puncture in ambulatory patients from 25.5 per cent to 13.5 per cent. The preliminary use of this drug also makes the lumbar puncture less difficult for the operator and less of an ordeal to the patient.

Neurologic Hazards of Spinal Anesthesia

As Smith[18] points out, it has been shown, both experimentally and clinically, that spinal anesthesia may be followed by neurological disorders, such as mild meningeal reactions, at times associated with an ocular palsy, more severe meningeal irritation with adhesions to the cord, and rarely degenerative lesions of the cord. Smith cites a case of typical hysterical anesthesia and paralysis of the legs, developing about six months after the anesthetic was administered. Another patient developed a schizophrenic syndrome following the anesthesia.

Of a group of forty-five patients subjected to spinal anesthesia, Martin and Halbron[19] observed neurological complications in eight cases. Four patients had headache, one transient urinary and fecal incontinence, one urinary retention for four days, and one extensive ocular paralysis and trigeminal anesthesia.

Hyslop[20] reports a case of "aseptic meningoencephalitis" following spinal anesthesia. He

recommends, as a possible preventive, drainage of spinal fluid after the operation, in order to remove any drug that may be not yet fixed in the tissues, or otherwise eliminated.

Linney[21] cites a fatal case following spinal anesthesia. Within a few minutes after the injection, the patient's fingers were numb and soon his speech became thick and muffled, which was followed by complete loss of speech. A few minutes later respiration ceased. Intravenous saline, glucose, and adrenalin proved futile.

Loeser[22] reports five cases of neuritis which developed from one to three weeks after the administration of the spinal anesthesia and which lasted a few months. In two cases, the ulnar nerve was involved; in one case the radial; and in the other two, the sciatic nerve.

Physiology and Pathology of the Cerebral Vessels

The Nerve Supply of the Cerebral Blood Vessels

It is Stöhr's[23] opinion that there are no specific vascular nerves in the brain. He states that there is anatomic evidence to show that although the entire vascular system is under nervous control, the syncytial character of the sympathetic nervous system makes it impossible to separate the nerves of the vessels from those of the surrounding tissue. To assume a special "vasoneurotic diathesis" is, according to Stöhr, taking a too narrow view, as the interlacing of nerve supply necessarily brings adjacent muscle, gland or other tissue under the same abnormal influence.

Although it has been demonstrated that the vessels of the brain are under nervous control, this does not prove, as Cobb[24] states, that normally this mechanism plays an important function. At present it can best be stated that there is a partial vasomotor control of the cerebral vessels.

Polycythemia and the Problem of Central Regulation of the Blood Picture

Salus[25] cites the following five cases as evidence of a central nervous regulation of the red corpuscles: (1) postencephalitic narcolepsy with increased red blood count during the intermittent attacks of narcolepsy; (2) genuine narcolepsy with polycythemia; (3) the same; (4) postencephalitic obesity with mild Parkinsonism and polycythemia; (5) sarcoma of left frontal lobe with interbrain and hypophyseal symptoms and polycythemia. In all these cases there was a proportionate increase in red cells, hemoglobin, and reticulocytes.

On the basis of animal experiments and blood examinations in twenty-eight cases of organic nervous diseases, Riccitelli[26] concludes that (1) there are, in the brain, regulatory centers not only for the chemical but for the morphologic constituents of the blood; (2) these centers are probably in the floors of the third and fourth ventricles; (3) they exert controlling influence not only on the peripheral blood but on the differentiated hematopoietic tissue.

Intracranial Hemorrhage and Purpura Hemorrhagica

After reviewing this subject, which shows that cerebral, meningeal, and ventricular hemorrhages have occurred in this disease, Alpers and Duane, Jr.[27] relate two new cases, one fatal but without necropsy. In the first case, a man forty-seven years old with a history of epistaxis, had choked disc and retinal hemorrhage; the further course is unknown. The second patient, a girl of four years of age, had a long history of purpuric spots in the skin and mucous membrane and intestinal hemorrhages. Toward the end disc swelling and retinal hemorrhages appeared; the spinal fluid was yellow and contained many red cells. After the third lumbar puncture a convulsion occurred. The temperature rapidly rose to 107 degrees and the child died.

Polycythemia and Its Neurologic Complications

Sloan[28] calls attention to the predominantly nervous and mental symptomatology recorded in the literature in this condition. He reports four cases. In the first, in addition to the usual features of polycythemia, thrombosis of the Sylvian artery with paresis of the lower right part of the face and right arm and various aphasic manifestations were presented. In addition, the patient had a spontaneous subarachnoid hemorrhage. The second, third, and fourth cases illustrate the various mental manifestations observed in polycythemia vera.

Winkelman and Burns[29] report two cases of polycythemia, in which mental confusion was prominent. The brain on necropsy was enlarged and purplish. Perivascular edema was marked and occasional perivascular hemorrhages were present. Severe degenerative changes in the ganglia cells were observed. A second patient showed a more marked psychosis; no necropsy was done.

Hypertensive Encephalopathy in Nephritis

Evans[30] stresses the point that the occurrence of such symptoms in nephritis as headache, convulsions, coma, and amaurosis often occur in the absence of uremia. He believes that these symptoms are most probably due to circulatory disturbances in the brain, resulting in ischemia and, in some cases, edema. The best remedies for hypertensive encephalopathy appear to be venesection, lumbar puncture, restriction of fluid intake, and morphine. It is Evans' belief that intravenous injection of hypertonic solutions to decrease intracranial pressure in these cases is

not without danger, since by thus increasing the blood volume even temporarily is hazardous.

Encephalitis

St. Louis Epidemic

According to Hempelmann[31], up to November 15, 1933 the total number of reported cases in St. Louis and that portion of St. Louis county immediately adjacent was 1,104 and the total number of deaths was 216, a mortality of 19.5 per cent. The following features characterized the epidemic: It occurred in the summer months and chiefly affected older adults, only about 11 per cent of the patients being less than fifteen years of age. Of 1087 cases analyzed, 58 per cent were of more than forty years of age, and 42.5 per cent more than fifty years of age. Second cases in the same family occurred in about 3.5 per cent. Clinical course: the onset was usually sudden with high fever and pronounced meningeal symptoms; recovery was ordinarily rapid and eye symptoms and sequelae were rare. Three types of cases could be distinguished:

Type I showed an abrupt onset with headache, high fever, nausea, stiff neck, and positive Kernig. Convulsions might occur. Mental confusion, aphasia, and tremor of hands, tongue and lips were common. Drowsiness might be supplanted by hyperexcitation but noisy delirium was rare. Pains in the back and limbs were rather frequent and occasionally some hyperesthesia occurred. Ocular manifestations were rare, the commonest being slight blurring of vision and less frequently transient double vision. Neurologically, the commoner signs were absence of abdominal reflexes, positive Kernig sign, and often pathological toe signs. Fundus examination showed only engorgement of retinal vessels. The pupils were small but reacted well. The temperature curve was usually highest at the onset of the encephalitic symptoms, falling by lysis and reaching normal in about six to ten days; occasionally it fell by crisis, or was unduly prolonged. Retention of urine was common, especially in older individuals. The leukocyte count varied from 4000 to 36000. Spinal fluid cell counts were between 300 and 500.

In type II a definite prodromal period of one to four or more days was present. This was characterized by headache, fever, grippy pains, sore throat, or other mild respiratory symptoms, photophobia, and mild conjunctivitis and chilly sensations. Usually the temperature fell during the next four days and the patients seemed to be on the road to recovery, when there would be a sudden exacerbation of the fever, and headache and a typical encephalitic picture developed.

Type III showed very mild symptoms—headache, fever, stiff neck were usually present but no tremors or mental confusion. Lumbar puncture showed an increase in cells. Recovery was rapid as a rule, often within ten days to two weeks. Pathologically, there was an intense vascular congestion, cellular infiltration, and a toxic degeneration of the nerve cells.

Other Forms of Encephalitis

Other types of encephalitis, including the more diffuse forms (encephalomyelitis, ophthalmo-encephalomyelitis, disseminated encephalomyelitis) following smallpox, chicken pox, measles, scarlet fever, whooping cough and mumps, continue to be reported, including those by Brouwer, de Jongh, and Rochat[32]; Van Bogaert[33]; Dubois, Ley, and Dagnelie[34]; Young[35]; McKaig and Woltman[36]; ter Braak and van Herwaarden[37]; and Stout and Karnosh[38]. The last-named authors analyzed twenty-eight cases of disseminated encephalomyelitis, occurring in epidemic form in Ohio. Of these, ten recovered; three were convalescing, six showing residual features; five relapsed; three died and one is unknown.

Twenty cases of encephalomyelitis are also reported from Cincinnati by McIntyre[39]. Death occurred in nine cases.

Sydenham's Chorea (Chorea Minor)

Wallace[40] analyzed 219 cases of this type of chorea. A high proportion of these cases had had rheumatism in the past. In some cases, certain physical and nervous factors other than rheumatism appeared to act as exciting causes of the chorea. The study also brought out that left-handed children appeared to be more liable to contract chorea than right-handed children. Involvement of the heart was present in 42.5 per cent of the total cases.

Treatment of Sydenham's Chorea

Bateman[41] reports good results from typhoid vaccine injections in this disease. The best effects were observed when the temperature was 104 to 105 degrees. Injections were given intravenously daily until the choreic movements disappeared. For greatest effectiveness, it was usually found that the vaccine must be doubled each day if an adequate fever was to be maintained. Most patients showed a marked improvement after two or three treatments and were usually free from choreic movements in a week.

Another therapeutic procedure for chorea minor is the use of nirvanol (phenylethyldantoin). Whitaker[42] discusses this treatment. In his experience, there is no danger from the use of this drug in properly controlled cases without complications. There is suggestive evidence that nirvanol may be of definite antirheumatic value. Furthermore, by shortening the course of the chorea, the incidence of subsequent carditis may be lessened.

Neurosyphilis

The Argyll-Robertson Pupil

Merritt and Moore[43], who analyzed the records of 749 cases of neurosyphilis, found that the Argyll-Robertson pupil was observed in 287 cases or 38.3 per cent. The cases were subdivided as follows: the tabetic or dementia paralytica forms of neurosyphilis 240 cases or 83 per cent; other forms of neurosyphilis forty-seven cases or 17 per cent. Merritt and Moore define the Argyll-Robertson pupil as one having the following characteristics: (a) an absence of the reaction to light; (b) miosis; (c) imperfect dilatation in response to installations of atropin and to painful stimuli; (d) an absence of reaction to vestibular stimulation; and (e) an active reaction on accommodation for near objects.

As a result of their studies, Merritt and Moore conclude that these characteristics are due to a destruction of the pupillary light reflex fibers and the sympathetic fibers. These fibers run together for a short distance in the anterior end of the brain stem just ventrally to the posterior commissure. A destructive lesion at this point would explain all of the phenomena of the Argyll-Robertson pupil. Any lesion at this point would produce the Argyll-Robertson pupil, but the only incontrovertible cases heretofore reported were associated with syphilis of the nervous system, except a few cases associated with gliomatous invasion in this region. Therefore, for all practical purposes, the Argyll-Robertson pupil is pathognomonic of syphilis of the central nervous system.

Treatment of Neurosyphilis

Wagner-Jauregg[44], who introduced the malarial treatment of general paresis, doubts the value of any fever producing agency except the production of an actual disease. He divides all fever producing agencies into three classes: (1) those not derived from microörganisms; they are the poorest; (2) products of microörganisms; (3) the infectious diseases themselves. He still finds tertian malaria the most useful.

Comparison of Various Forms of Fever Therapy

Wilgus and Kuhns[45], who examined the records of 500 general paretics treated by various forms of fever therapy, gives the following table of results:

Forms of Therapy	Improved	Unimproved	Worse
Typhoid vaccine	52%	28%	20%
Sulphur in oil	58%	21%	21%
Malarial therapy	66%	20%	14%
Diathermy	72%	11%	17%
Electric blanket	78%	15%	7%

Diathermy Treatment in General Paresis

Freeman, Fong, and Rosenberg[46] are very pessimistic regarding the value of this form of therapy in general paresis. In their hands, it met with almost complete failure. They believe that the early reports on the diathermy treatment were published after too short a period of observation. Malaria, in their experience, is by far the better form of fever therapy.

Their experience agrees with that of Worthing[47]. He states that although there is some place for diathermy in the treatment of the more coöperative patients, there is actually more suffering with diathermy than with malaria.

Hyperthermia Treatment by Means of Hot Baths

Walinski[48] has treated 353 patients with neurosyphilis by this method during the past five years. He first administers 10 cc. of a 20 per cent salt solution to diminish sweating and strengthen the heart. The patient is then placed in a tub of water of 38 degrees centigrade, the temperature of which is raised in twelve to thirty minutes to 41 to 42 degrees. The desired body temperature is then usually reached, and the patient is placed in a dry, hot pack and given hot drinks. Severe heart and kidney disease are contraindications to the treatment. In ninety cases of tabes so treated, surprisingly good results were obtained, particularly as regards gastric crises and lancinating pains. Of sixteen cases of general paresis, complete remission took place in 19 per cent, and in another 30 per cent there was improvement so that the patients could do some work.

Tryparsamide Treatment of Neurosyphilis

Reese[49], who analyzes tryparsamide and malarial therapy in neurosyphilis, states that of 341 cases of general paresis treated by the former drug, there were 54 per cent showing clinical arrest or remissions and 78 per cent showing clinical or serological cures. According to Reese, a survey of the treatment of general paresis with tryparsamide or malaria shows much the same percentage of remissions or improvement. The best results are probably obtained from the combined use of tryparsamide and malaria. He usually begins with the former and decides later which is the better form of therapy.

The Spinal Cord

Pathogenesis of Poliomyelitis

Simon Flexner[50] states that the evidence is now strong that the virus of this disease ascends from the nasal membranes to the olfactory lobes of the brain, and then continuously by nerve conduction to the mid-brain and spinal cord. He further states that since the virus, as originally present in human nervous tissues, is of low infectivity for monkeys, the failure to induce disease in these animals by inoculation of spinal fluid is not conclusive evidence of its absence from the spinal fluid. The virus em-

ployed for the experimental nasal installation is highly potent for monkeys. Hence the altered spinal fluid withdrawn from the animals so inoculated was injected intravenously into the Macacus rhesus monkeys, and the procedure was followed by an accelerating injection eight days later. No detectible pathologic effects arose in these animals. Therefore, Flexner concludes that in man and the monkey, the virus of poliomyelitis, even in small amounts, does not pass from the infected nervous tissue into the cerebrospinal fluid; and the cellular changes in the fluid represent a reaction of the nervous system to injury and are not the result of the virus in the fluid itself.

Forced Drainage in the Treatment of Poliomyelitis

This method of therapy was worked out by Kubie and has been used in several diseases of the nervous system, including poliomyelitis. Kubie and Retan[51] make a recent report on their experience with this form of treatment. By forced drainage is meant the washing out, so to speak, of the ventricles and the subarachnoid spaces, including the perivascular and pericellular spaces of the brain and cord. This is accomplished by drinking large amounts of water and, at the same time, draining the spinal fluid for several hours to a few days. The authors explain that it is only under the special conditions of forced drainage that the formation of cerebrospinal fluid in large amounts throughout all the parenchymatous tissue of the central nervous system can occur. Pure drainage alone draws only the surface fluid of the pre-existing lake of cerebrospinal fluid and obstructs the formation of any fluid and also drainage from the depths by allowing collapse of the leptomeninges. Forced drainage causes fluid from the perivascular channels and the depths to course to the surface, carrying with it any products of inflammation. Under such conditions, there is no increase in intracranial pressure or any hydration of the parenchyma. This type of treatment was tried in different types of syphilis of the nervous system, multiple sclerosis, epidemic encephalitis, Parkinson's disease, and chorea. Although there are at present not enough cases to make statistical treatment possible, the clear-cut symptomatic and clinical changes after forced drainage shows that this therapy affects the course of acute and chronic infections of the nervous system. The authors point out surprising results in chronic cases of syphilis. Alterations were observed in reflexes; changes were noted in the pupillary responses; disappearance of chronic and intractable pain, and improvement in sphincteric control.

Polyganglionitis (Posterior Poliomyelitis)

An interesting example of this uncommon infection is reported by von Sántha[52]. The patient, sixty years of age, began to complain in 1931 of backache, headache, and fatigue. The following year his gait was weak and unsteady. The knee and ankle jerks on the left were absent and sluggish on the right side. The spinal fluid showed marked increase in protein and gave a positive Lange test in the higher dilutions. The ataxia increased and all tendon reflexes and the abdominal and cremasteric reflexes disappeared. Sensation was much impaired in the trigeminal area and elsewhere. The patient died in 1933. Necropsy revealed degeneration of the posterior columns of the cord and thinning of the posterior roots. The peripheral nerves showed secondary degeneration. Microscopically, the peripheral nerves of the upper and lower extremities contained both severely degenerated and perfectly normal fiber bundles. In all the spinal ganglia examined, most of the cells had been destroyed. All the posterior roots were almost completely demyelinated. The anterior roots were normal. The posterior columns of the cord showed degeneration up to the nuclei in the bulb. The gray matter in the thoracic region showed large lymphocyte infiltrations about the veins. All parts of the gray matter in the lower thoracic region were affected. There was fatty degeneration of the root fibers of the trigeminal and glossopharyngeal nerves and the tractus solitarius. The cerebral cortex and basal ganglia were practically normal.

Antirabic Vaccine Paralysis

Three fatal cases of Landry's type of ascending paralysis and two cases of transverse cord lesions with recovery are reported by Stuart and Krikorian[53]. A carbolized vaccine had been used in the treatment of these cases of rabies.

After giving a detailed description of these cases and of another case of Landry's paralysis without antirabic vaccine, Getzowa, Stuart, and Krikorian[54] conclude that there seems to exist some deleterious substance in the antirabic vaccine, capable in peculiarly susceptible individuals of producing neuroparalytic disorders. The predominant feature in these cases of Landry's paralysis is a widespread lesion of ganglia cells, inducing the occurrence of a rapidly advancing and rapidly fatal paralysis. Because of the total absence of perivascular zones of demyelization and of perivascular cuffing, it is probable that the antirabic treatment factor does not belong in the heterogenous group of factors capable of producing acute disseminating encephalomyelitis.

Fatal Ascending Paralysis Following Typhoid Vaccination

The meager literature on the nervous sequelae to typhoid vaccination is reviewed by Gayle, Jr., and Bowen[55]. These authors report an unusual reaction following typhoid vaccination. Their patient, a few days after inoculation with

a .5 cc. typhoid-paratyphoid vaccine, complained of numbness in his arms and legs. After a second injection of 1 cc. two weeks later, he gradually developed weakness in the legs and arms. Three weeks later he had flaccid paralysis, complete in the legs, and partial in the arms. The tendon reflexes and abdominal reflexes were absent, and there was diminished vibratory sensation. Within a few days, fatal bulbar paralysis set in. Postmortem examination of the cord showed swelling and degeneration of the cells in the cord and bulb. The peripheral nerves near their exit from the spine were described as swollen, congested, and degenerated.

Pathogenesis and Treatment of Multiple Sclerosis

Putnam[56] has been able to obtain local areas of demyelinization imitating the plaques of multiple sclerosis by injecting tetanus bacilli, by inducing carbon monoxide poisoning, and by injecting emulsions of cod-liver oil into the carotid artery of cats and bland oily substances into the ligated longitudinal sinuses of dogs. Putnam states that since the lesion is produced by a simple mechanical interference with the blood supply, it is superfluous to postulate a specific demyelinizing organism, virus, or toxin to account for the pathological picture. The ultimate etiologic factor in multiple sclerosis should probably be sought in the local vascular abnormality or in some alteration in coagulability of the blood. As regards therapy in multiple sclerosis, Putnam states there have been no positive advances made. Most of the empirical methods of treatment, including fever therapy, arsenicals, antimony compounds, high vitamin diet, x-ray treatment, ultraviolet light, forced drainage of the spinal fluid, and quinine have been thoroughly tried without more success than can be accounted for by the hope which they engender in the patient's mind.

Lead as a Possible Cause of Multiple Sclerosis

Cone, Russel, and Harwood[57] have made an investigation of the occurrence of lead in multiple sclerosis. This substance was found in the spinal cord at necropsy in a typical case of multiple sclerosis. In another case of neuromyelitis optica, lead was present in the brain and in the spinal cord. The brain contained larger amounts of lead than the normal-appearing brain. The authors were also able to demonstrate lead in the stools, urine, and cerebrospinal fluid of six cases of the disease. In the case of neuromyelitis optica, calcium stopped the advancing myelitis and caused lead to disappear from the spinal fluid. The authors state, however, that it is too early to state whether calcium is a satisfactory therapeutic agent in multiple sclerosis. They do not dogmatically state that because lead is found in the central nervous system, in the spinal fluid, in the bones, and liver, and in the excreta, it is, therefore, the cause of the disease, although the constant association of the metal in their cases of multiple sclerosis strongly incriminates it. They finally conclude that their work suggests lead as a possible etiologic agent in multiple sclerosis of the exacerbating and remitting type.

Treatment of Cord Degeneration in Pernicious Anemia

Meulengracht's[58] experience with 136 cases of pernicious anemia, observed since he started liver therapy in 1927, brings out the following striking features: (1) many patients developed cord symptoms during liver treatment; (2) there was much evidence to show that these patients had not received sufficient liver, and that patients with cord changes required larger doses than patients without cord symptoms. The author found it gratifying that the cord symptoms frequently subsided by increasing the amount of liver or by adding or substituting ventriculin.

Peripheral Nerves

Alcoholic Neuritis

Minot, Strauss, and Cobb[59] take up the problem of dietary deficiency as a factor in the production of this form of neuritis. After studying fifty-seven cases and the records of seventy-three additional cases, the authors conclude that dietary deficiency, probably particularly the lack of vitamin B-1, plays an important part in the production of alcoholic polyneuritis. Furthermore, it is probable that the deficiency is attributable not only to inadequate intake, but also to the state of the gastrointestinal tract and the presence of factors that can inhibit the effectiveness of nutritional elements.

Neurological Complications of Serum Sickness

Doyle[60] reports one, a case of complete flaccid paralysis of the deltoid and partial of the biceps and some shoulder muscles which came on seven days after prophylactic injection of scarlet fever antitoxin and, two, a case of similar paralysis following injection of tetanus antitoxin. A review of the literature made by Doyle disclosed forty-nine cases of neurologic complications from serum injections; thirty-nine followed tetanus antitoxin; seven diphtheria antitoxin; five scarlet fever antitoxin; two antipneumococcic serum type I; and one antimeningococcic serum. In most of these cases, the neurological complication was an involvement of some part of the brachial plexus.

Etiology and Treatment of Retrobulbar Neuritis

From a review of 225 cases of retrobulbar neuritis at the Mayo Clinic, Benedict[61] gives the following data as to etiology: multiple sclerosis in 155 cases; pernicious anemia and nicotine in

fourteen; diabetes in fourteen; alcohol and tobacco in twenty-eight; syphilis in two; congenital amblyopia in four; familial causes in one; sinus disease in one; postpartum hemorrhage in one; plumbism in two; and indeterminate causes in three cases. In more than 500 definitely proved cases of multiple sclerosis seen at the clinic, disturbance in vision was given as the first symptom in about fifteen per cent. In another thirty-five to forty per cent, disturbances of vision were seen in subsequent episodes. In all the cases, the syndrome of retrobulbar neuritis was the symptom most commonly observed. Intranasal sinus operations had been performed elsewhere in more than sixty per cent because of visual disturbances. The treatment found most successful in the author's hands has been intravenous injections of triple typhoid vaccine in increasing doses, from 25,000,000 to 450,000,000 bacteria, the injections being given three to four times weekly until the vision has returned, or until it becomes evident that the treatment is of no avail.

Roentgen Treatment of Trigeminal Neuralgia

In Hummel's[62] experience with twenty-seven cases of trigeminal neuralgia by x-ray, good results were obtained in 37 per cent. The treatment is directed at the gasserian ganglia, from one to fourteen treatments being given at intervals of eight days.

Surgical Treatment of Meniere's Disease

Equally encouraging as Dandy's results reported in 1928, are Coleman's and Lyerly's[63] results in the surgical treatment of Meniere's disease. The latter report ten cases in which intracranial section of the eighth nerve for the relief of that condition was done. The operation was done with a negligible risk and recovery from the operation was prompt in every case. The diagnosis of Meniere's Disease is based on the history of attacks of violent vertigo, accompanied by nausea and vomiting, tinnitus in one ear and partial deafness in the same ear. Although following the operation some of the patients have had slight unsteadiness, particularly on sudden change of position, none of them have had an attack of vertigo. The unsteadiness tends to improve with time and is not disabling. Tinnitus, when not entirely abolished by the operation, has been improved in every case. In the discussion of these results, Dandy states that in his experience with thirty cases of Meniere's disease on which he has operated, there was no mortality and no return of attacks of vertigo; there was no loss of function except the remaining hearing which is of no practical value.

Treatment of Sciatica

Craig and Ghormley[64] report their therapeutic results in sciatica due to various causes: in eighty cases epidural injection caused complete relief in 52 per cent, marked relief in 24 per cent, and no relief in 22 per cent. In thirty-six cases diathermy caused complete relief in 33 per cent, moderate relief in 12 per cent, and no relief in 55 per cent. The combination of epidural injection and diathermy was applied in 21 per cent. In 42 per cent of these, there was complete relief; in 10 per cent moderate relief, and in 48 per cent no relief. A sacroiliac belt and diathermy were employed in fifty-two cases. Relief was complete in 32.6 per cent; moderate in 13 per cent, and in 54.4 per cent there was no relief. Epidural injection, a belt, and diathermy were tried in eight cases. Eighty-five per cent of these patients were completely relieved, 2 per cent moderately relieved, and 13 per cent were not benefited. On twenty-eight patients who were confined to bed, the following measures were employed: double Buck's extension, diathermy, epidural injection, intravenous injection of foreign protein, and elimination of foci of infection. Of these patients 85.7 per cent were completely relieved, 14.3 per cent moderately relieved. In fourteen cases the same measures were employed except that the epidural injections were omitted. Complete relief resulted in 63 per cent, moderate relief in 23 per cent, and no benefit in 14 per cent.

Vegetative Nervous System

Many papers dealing with the results of sympathectomy for various conditions have been written in the past year. The following are chosen for brief reviews:

Resection of Splanchnic for Essential Hypertension

Craig and Brown[65] make a preliminary report of four cases of hypertension, in which the splanchnic nerves were resected. The results showed some reduction in the levels and responses of the systemic blood pressure, except in the most severe cases.

Treatment of Volkmann's Contracture by Periarterial Sympathectomy

Landivar and Iparraguirre[66] report the case of a woman, aged twenty-one, who developed a Volkmann's contracture following fracture of the distal end of the radius. Humeral arteriectomy for a length of 10 cm. was followed by complete disappearance of the contracture. The patient obtained complete functional recovery and the appearance of the hand and arm was normal two years after the operation.

Sympathectomy for Relief of Periodic Edema of Hand

Abbott[67] reports a case of a Jewish woman, thirty years old, in whom intermittent swelling and discoloration of the entire left hand had persisted for eighteen years. In September of 1931 a cervicothoracic lumbar sympathectomy

was performed. Since then the hand has been normal.

Sympathectomy for Raynaud's Disease

Gask[68] obtained good immediate results in seven cases in which he excised a portion, one inch long, of the thoracic sympathetic trunk from below the second thoracic ganglion up to and including the stellate ganglion. Although the immediate results of the treatment are good, states the author, the improvement is not lasting in all cases, some remaining greatly improved and others only slightly.

Resection of Perisacral Nerve for Bladder Condition

The results at the Mayo Clinic with sympathetic neurectomy are reported by Adson[69]. Eight cases were operated on for paresis of the musculature of the bladder due to injury. Two were considered cured and four improved. In two cases in which there was difficulty in starting the flow of urine, sympathetic neurectomy resulted in immediate and continuous cure of the difficulty. In six of the eleven cases with inveterate vesicle pain caused by various conditions, the results after presacral neurectomy have been satisfactory.

The Endocrines

Diagnosis and Treatment of Hypogonadism in the Male

McCullagh, McCullagh, and Hicken[70] have isolated a testicular hormone called "androtin" which appears to be effective in the treatment of certain cases of sexual impotence in the male. These authors have made an assay of 200 specimens of body fluids for testicular hormone by means of a capon method. They report the results of treatment with androtin in seven cases of hypogonadism. They found that the most demonstrable changes following treatment seemed to be a common tendency (1) to a rise in basal metabolism, (2) to a fall in the level of blood cholesterol, and (3) to recovery of testicular hormone from the urine after its administration. In some cases motile spermatozoa have been found in fluid obtained by prostatic massage after the treatment, where none were found before. Common symptomatic changes following the treatment have been the following: increase in sexual potency, decrease in vasomotor and nervous disturbance, increase in frequency of nocturnal emissions, and increased energy.

Therapeutic Use of Adrenal Cortex Hormone

Leyton[71] has tried intravenous injections of an active extract of suprarenal cortex in selected cases of neurasthenia. The suitable cases seemed to be those which followed some infection, cases with low blood pressure, low blood sugar, and subnormal temperature. Leyton found the results sufficiently promising to justify a therapeutic trial on a wider scale.

Robbins[72] observed excellent results from the use of the extract in an asthenic condition following an acute streptococcic sore throat with a low blood pressure.

Hartman, Beck, and Thorn[73] describe three cases in whom the main complaint was nervous or muscular fatigue. In the first case, a male, aged forty-seven, who had been complaining of fatigue, insomnia, visual disturbance at night, tremor, and pyloric spasm since 1926, was greatly benefited by injections of cortin. The second case, a female, aged twenty-seven, who had lost three pounds in the last three years and who since February, 1931 had been complaining of periods of weakness, during which anorexia, restless sleep, visual difficulty, palpitation, dyspnea, irritability, and depression were prominent, felt well when put on 160 Gm. of cortex daily, but when reduced to 120 Gm. or less showed a return of symptoms. Case three was a male, aged forty-one, who had had typical anxiety attacks for a year. He showed physical signs of marked nervous tension; blood pressure was 135 systolic and 90 diastolic. He improved considerably on cortin, but it was impossible to continue the treatment as long as seemed desirable.

Muscular Dystrophy and Myasthenia Gravis

Tripoli and Beard[74] report their clinical and biochemical results following the oral administration of amino acids. These authors state that the results of many studies have shown that in cases of muscular dystrophy, the creatine metabolism of the muscles becomes deranged. It is well known from the work of many investigators that the patient suffering from muscular dystrophy is unable to retain creatine in the muscles. When ingested, most of it is excreted in the urine, making the patient practically "diabetic" as regards creatine. This is accompanied by a great loss of muscle tissue and function. When creatine is given as such, most of it passes through the body and is excreted in the urine. Thus, feeding creatine is useless in the treatment of muscular dystrophy. On the other hand, the feeding of amino acids, such as glycine and glutamic acid, cause for the first few weeks an increased creatinuria which gradually subsides, the creatine being retained in the muscles. At this time clinical improvement and an increase in function is noted.

Tripoli and Beard administered glycine or glutamic acid, fed in 10 or 20 Gm. doses daily in six cases of various dystrophies and atrophies. The total number of cases reported by these authors and others to date is sixty-nine, fifty-one of which showed decided clinical improvement after amino acid therapy. The most marked clinical improvement is noted in those cases

grouped as muscular dystrophies (caused by primary lesions in the muscle which probably are primarily abiotrophic). Very little improvement was noted in cases grouped as progressive nuclear muscular atrophy.

Boothby[75] makes his third report on the results obtained by the use of glycine and ephedrin in myasthenia gravis observed at the Mayo Clinic. The glycine is usually given in doses of 30 Gm. daily, together with, at times, 3/8 grain of ephedrin twice daily. Twelve patients were treated. Ten showed definite improvement, four of whom were markedly improved. In two there was no response to treatment except that the progress of the disease was apparently arrested. One of these two died from causes not directly attributable to the myasthenic syndrome. Boothby finally concludes that by the careful use of either ephedrin or glycine, more often of both, most patients with myasthenia gravis can be improved sufficiently to permit them to return to work or, at least, to enjoy a useful life. Time alone will tell whether this improvement can be maintained.

REFERENCES

1 Saul, L. J., and Davis, H.: Action currents in the central nervous system. Arch. Neurol. and Psychiat. 29:255 (Feb.) 1933.

2 Adrian, E. D.: The activity of nerve-cells. Lancet. 2:664 (Sept. 16) 1933.

3 Foerster, O.: The dermatomes in man. Brain. 56:1 (March) 1933.

4 Penfield, W.: Evidence for a cerebral vascular mechanism in epilepsy. Ann. Int. Med. 7:303 (Sept.) 1933.

5 Lennox, W. G.: The multiple causes of seizures in the individual epileptic patients. New Eng. J. Med. 209:386 (Aug. 24) 1933.

6 Fremont-Smith, F., and Merritt, H. H.: Influence of variations in fluid intake on intracranial pressure in epileptics. Arch. Neurol. and Psychiat. 29:454 (March) 1933.

7 Lyon, D. M., and Dunlop, D. M.: Epilepsy and dehydration. Edinburgh M. J. 40:266 (May) 1933.

8 Fetterman, J. L., and Kumin, H. J.: Dehydration in epilepsy. J. A. M. A. 100:1005 (April 1) 1933.

9 Storchheim, F.: Status epilepticus treated by magnesium sulphate, injected intravenously. J. A. M. A. 101:1313 (Oct. 21) 1933.

10 Riley, H. A., Brickner, R. M., and Kurzrok, R.: The abnormal excretion of theelin and prolan in patients suffering from migraine. Bull. Neurol. Inst. New York. 3:53 (June) 1933.

11 Critchley, M., and Ferguson, F. R.: Migraine. Lancet. 1:123 (Jan. 21) 1933 and 1:182 (Jan. 28) 1933.

12 Hahn, L.: Zur Gegässtheorie der Migraine. Med. Klin. 28:1264 (Sept. 9) 1933. (See also Fischer-Wasels, B.: Die funktionellen Störungen des peripheren Kreislaufs. Frankfurt. Ztschr. f. Path. 45:1, 1933.

13 Crandall, L. A., Jr., and Roberts, G. M.: Treatment of periodic headache with chondroitin sulphuric acid. Illinois M. J. 63:513 (June) 1933.

14 Kottmann, K.: Treatment of migraine with gynergen. Schweiz. med. Wchnschr. 63:572 (June 10) 1933.

15 Flexner, L. B.: The rôle of ventricular dilatation in the spread of infections from the subarachnoid space into ventricles. Bull. Johns Hopkins Hosp. 53:140 (Sept.) 1933.

16 Fremont-Smith, F., and Merritt, H. H.: Relationship of arterial blood pressure to cerebrospinal fluid pressure in man. Arch. Neurol. and Psychiat. 30:1309 (Dec.) 1933.

17 Kulchar, G. V., and King, A. D.: Use of sodium amytal in prevention of reactions associated with lumbar puncture. Arch. Neurol. and Psychiat. 30:170 (July) 1933.

18 Smith, W. A.: Neurologic hazards of spinal anesthesia. J. M. A. Georgia. 22:297 (Aug.) 1933.

19 Martin, R. H., and Halbron, P.: A propos des accidents de la rachi-anesthésie. Paris méd. 23:217 (March 11) 1933.

20 Hyslop, G. H.: Spinal anaesthesia, nervous system sequelae; case in point. Surg. Gynec. Obst. 57:799 (Dec.) 1933.

21 Linney, R. Z.: Spinal anesthesia. South. Med. and Surg. 95:312 (June) 1933.

22 Loeser, L. H.: Peripheral neuritis as a sequela of spinal anesthesia. J. A. M. A. 101:31 (July 1) 1933.

23 Stöhr, P., Jr.: The nerve supply of blood vessels. Deutsche med. Wchnschr. 59:1625 (Oct. 27) 1933.

24 Cobb, S.: The cerebral circulation. XXV. Remarks on clinical physiology. Collected Papers. Dept. of Diseases of the Nervous System. Harvard Medical School, Boston, 1934.

25 Salus, F.: Polycythemia and problem of central regulation of blood picture. Deutsches Arch. f. klin. Med. 175:214, 1933.

26 Riccitelli, L.: Über die Regelung der morphologischen Blutelemente seitens der Nervenzentren. Deutsche med. Wchnschr., 59:1395 (Sept. 8) 1933.

27 Alpers, B. J., and Duane, W., Jr.: Intracranial hemorrhage in purpura hemorrhagica. J. Nerv. and Ment. Dis. 78:260 (Sept.) 1933.

28 Sloan, L. H.: Polycythemia rubra vera: neurologic complications; report of four cases. Arch. Neurol. and Psychiat. 30:154 (July) 1933.

29 Winkelman, N. W., and Burns, M. A.: Polycythemia vera and its neuropsychiatric features. J. Nerv. and Ment. Dis. 78:597 (Dec.) 1933.

30 Evans, H.: Hypertensive encephalopathy in nephritis. Lancet. 2:583 (Sept. 9) 1933.

31 Hempelmann, T. C.: Encephalitis—the St. Louis epidemic. Chicago Med. Soc. Bull. Dec. 2, 1933.

32 Brouwer, B., de Jongh, C. L., and Rochat, R. R.: Über die Veränderungen im Zentralnervensystem bei Pocken (Variola). Deutsche Ztschr. f. Nervenh. 131:1, 1933.

33 Van Bogaert, L.: Nervous sequelae of chicken-pox, measles and scarlet fever. Rev. neurol. 1:150 (Feb.) 1933.

34 Dubois, R., Ley, R.-A., and Dagnelie, J.: Whooping cough encephalitis. Rev. franc. de pediat. 9:49, 1933.

35 Young, R. C.: Mumps encephalitis. New Orleans M. and S. J. 86:25 (July) 1933.

36 McKaig, C. B., and Woltman, H. W.: Neurologic complications of epidemic parotitis; report of case of parotitic myelitis. Proc. Staff Meet. Mayo Clin. 8:542 (Sept. 6) 1933.

37 ter Braak, J. W. G., and van Herwaarden, A.: Ophthalmoencephalo-myelitis. Klin. Monatsbl. f. Augenh. 91:316 (Sept.) 1933.

38 Stout, R. E., and Karnosh, L. J.: Acute disseminated encephalomyelitis. J. A. M. A. 101:667 (Aug. 26) 1933.

39 McIntyre, H. D.: An unusual encephalopathy, probably infectious in origin. J. A. M. A. 100:1097 (April 8) 1933.

40 Wallace, H. L.: Chorea. Edinburgh M. J. 40:417 (Sept.) 1933.

41 Bateman, D.: A treatment of Sydenham's chorea. Brit. M. J. 1:1003 (June 10) 1933.

42 Whitaker, W. M.: Recent developments in treatment of chorea minor. Illinois M. J. 63:123 (Feb.) 1933.

43 Merritt, H. H., and Moore, Merrill: The Argyll Robertson pupil; an anatomic-physiologic explanation of the phenomenon, with a survey of its occurrence in neurosyphilis. Arch. Neurol. and Psychiat. 30:357 (Aug.) 1933.

44 Wagner-Jauregg, J.: Fever treatment of general paresis. Wien. med. Wchnschr. 83:16 (No. 1), 1933.

45 Wilgus, S. D., and Kuhns, R. H.: Study of fever producing agents for treatment of general paresis. Arch. Phys. Ther. 14:455 (Aug.) 1933.

46 Freeman, W., Fong, T. C., and Rosenberg, S. J.: Diathermy treatment of dementia paralytica. J. A. M. A. 100:1749 (June 3) 1933.

47 Worthing, H. J.: Diathermy in treatment of general paralysis. Psychiat. Quart. 7:245 (April) 1933.

48 Walinski, F.: Hyperthermia treatment by means of hot baths. Deutsche med. Wchnschr. 59:397 (March 17) 1933.

49 Reese, H. H.: Tryparsamide in treatment of neurosyphilis. J. Nerv. and Ment. Dis. 78:354 (Oct.) 1933.

50 Flexner, Simon: Early changes in cerebrospinal fluid of monkeys nasally instilled with virus of poliomyelitis. Science. 78:129 (Aug. 11) 1933.

51 Kubie, L. S., and Retan, G. M.: Forced drainage of cerebrospinal fluid in treatment of diseases of central nervous system. J. Nerv. and Ment. Dis. 77:56 (Jan.) 1933.

52 von Sántha, Kalman: Polyganglionitis (posterior poliomyelitis) with clinical picture of subacute pseudotabes. Arch. f. Psychiat. 100:398, 1933.

53 Stuart, G., and Krikorian, K. S.: Neuroparalytic accidents complicating antirabic treatment. Brit. M. J. 1:501 (March 25) 1933.

54 Getzowa, S., Stuart, G., and Krikorian, K. S.: Pathological changes observed in paralysis of Landry type. J. Path. and Bact. 37:483 (Nov.) 1933.

55 Gayle, R. F., Jr., and Bowen, R. A.: Acute ascending myelitis following the administration of typhoid vaccine. J. Nerv. and Ment. Dis. 78:221 (Sept.) 1933.

56 Putnam, T. J.: Pathogenesis of multiple sclerosis: possible vascular factor. New Eng. J. Med. 209:786 (Oct. 19) 1933.

57 Cone, W., Russel, C., and Harwood, R.: Lead as a possible cause of multiple sclerosis. Tr. Am. Neurol. A., 1933, p. 113.

58 Meulengracht, E.: Treatment of cord degeneration in pernicious anemia. Klin. Wchnschr. 12:1163 (July 29) 1933.

59 Minot, G. R., Strauss, M. B., and Cobb, S.: "Alcoholic" polyneuritis; dietary deficiency as a factor in its productions. New Eng. J. Med. 208:24 (June 15) 1933.

60 Doyle, J. B.: Neurologic complications of serum sickness. Am. J. M. Sc. 185:484 (April) 1933.

61 Benedict, W. L.: Retrobulbar neuritis and disease of nasal accessory sinuses. Proc. Staff Meet. Mayo Clin. 8:147 (March 8) 1933.

62 Hummel, R.: Roentgen treatment of trigeminal neuralgia. Strahlentherapie. 47:684, 1933.

63 Coleman, C. C., and Lyerly, J. G.: Ménière's disease; diagnosis and treatment. Arch. Neurol. and Psychiat. 29:522 (March) 1933.

64 Craig, W. M., and Ghormley, R. K.: Significance and treatment of sciatic pain. J. A. M. A. 100:1143 (April 15) 1933.

65 Craig, W. M., and Brown, G .E.: Resection of splanchnic nerves in cases of hypertension. Proc. Staff Meet. Mayo Clin. 8:373 (June 14) 1933.

66 Landivar, A. F., and Iparraguirre, L.: Volkmann's contracture treated by periarterial sympathectomy. Dia Med. 6:149 (Sept. 11) 1933.

67 Abbott, W. D.: Periodic edema of hand with seven day cycle: treatment with sympathetic ganglionectomy. J. A. M. A. 100:1328 (April 29) 1933.

68 Gask, G. E.: The surgery of the sympathetic nervous system. Brit. J. Surg. 21:113 (July) 1933.

69 Adson, A. W.: Results of sympathectomy in treatment of peripheral vascular diseases, Hirschsprung's disease, and cord bladder. Ann. Int. Med. 6:1044 (Feb.) 1933.

70 McCullagh, E. P., McCullagh, D. R., and Hicken, N. F.: Diagnosis and treatment of hypogonadism in the male. Endocrinology. 17:49 (Jan.-Feb.) 1933.

71 Leyton, O.: The use of extract of suprarenal cortex in one type of neurasthenia. Practitioner. 130:466 (April) 1933.

72 Robbins, J. H.: Use of cortin in case of acute hyposuprarenalism occurring as sequela of acute streptococcic sore throat. J. A. M. A. 100:657 (March 4) 1933.

73 Hartman, F. A., Beck, G. M., and Thorn, G. W.: Improvement in nervous and mental states under cortin therapy. J. Nerv. and Ment. Dis. 77:1 (Jan.) 1933.

74 Tripoli, C. C., and Beard, H. H.: Muscular dystrophy and atrophy; clinical and biochemical results following the oral administration of amino-acids. Arch. Int. Med. 53:435 (March) 1934.

75 Boothby, W. M.: Myasthenia gravis; the effect of treatment with glycine and ephedrin; third report. Arch. Int. Med. 53:30 (Jan.) 1934.

THE PHYSICIAN AND THE NURSE

1. Let us recognize the essential "team" relationship between the doctor and the visiting or public health nurse, each with a primary obligation to the patient, and with supplementary community obligations as well.

2. Let us try to resolve the actual or potential conflict between the private doctor and the treatment motive on the one hand, and the public health nurse and prevention on the other, by:

a. Retaining at least a minimum of the genuine personal service element in the field program of the nurse—a phase of her work which the doctor automatically understands and appreciates.

b. Utilizing the help of the nurse to expand for the doctor the growing field for the private practice of preventive medicine.

c. More fully informing the doctor as to the wide range of concrete services the nurse has to offer to him and his patient, in both treatment and prevention, perhaps using pneumonia and the communicable diseases as fresh wedges in more widely opening this field, particularly as it encompasses the non-indigent patient.

3. Let us avoid the errors of the past, recognizing the basic obligation of the nurse to uphold the procedures and standards of private medical practice, and to aid in preserving the personal relationship of doctor and patient.

4. Let us recognize the at least contemporary distinction in the doctor's mind between communicable diseases with their community and epidemiological relationships on the one hand, and the non-communicable, constitutional affections, with their essentially private features, on the other.

5. And finally, let us call upon our visiting nurse associations to furnish, in coöperation with other community agencies, the vision and leadership essential, first, to the acquainting of the doctor with the character and purpose of the visiting nurse association and the public health nurse; to the "selling" of the nurse's wide range of services to the doctor; to the wiping out of existing points of irritation and conflict; to the featuring of the doctor's and the nurse's common interests and objectives in both treatment and prevention as they affect the patient and the community. — Summary of an address by Donald B. Armstrong, M.D. Reprinted from *Public Health Nursing*, November, 1934.

EFFECT OF THE NON-RESIDENT ON DEATH AND BIRTH RATES IN BUFFALO

A cursory glance over the birth and death statistics for the city of Buffalo during past years reveals that a large proportion of such births and deaths are of persons residing elsewhere than in the city. During the first nine months of the current year, twelve per cent, or nearly one in eight deaths, and thirteen per cent, or more than one in eight births, were of such non-residents; and during the past five years these percentages have remained nearly constant. The result of these additions is to raise the rates by corresponding proportions, giving a picture of the vital conditions which is far from representative of the true status.

The Division of Vital Statistics of the New York State Department of Health has long recognized the difficulties involved in this situation. In 1926, in the Annual Report of that division, we read: "One of the most involved problems in the statistical evaluation of health conditions of communities is presented by the need of an equitable allocation of deaths of non-residents. The importance of considering the element of residence in computing death rates is becoming more evident each year. The growth of the 'hospital habit' which brings large numbers of sick people from rural and from small urban districts to well-equipped city hospitals for treatment is, in many instances, responsible for most or all of the disparity in the death rates of these sections of the State. Cities with excellent hospital facilities are likely to show, because of that fact, higher death rates than places less fortunate in this respect. At the same time, the death rates of certain rural districts are affected adversely by the existence within their limits of hospitals, sanatoria, and other institutions, most of whose inmates are not residents of the district."—*Sanitary Bulletin.* Published by the Buffalo Department of Health.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21011

PRESENTATION OF CASE

A seventy-one year old American widower was admitted to the Emergency Ward complaining of severe weakness.

An adequate history could not be obtained. He did say, however, that he had had severe weakness and vague abdominal pain of about six weeks' duration.

Physical examination showed a thin, emaciated man who was somewhat flighty and confused. He had evidently lost a large amount of weight. The tongue was dry, beefy and fissured. The fundi showed marked sclerosis. His lungs were clear except for a few râles at the right base. The heart was enlarged to the left, the apex beat being felt in the axillary line. The rate was regular and the beat forceful. There was a loud, high-pitched systolic murmur heard best at the apex and aortic area and poorly transmitted to the axilla. The blood pressure was 144/100. The brachial arteries were sclerotic and tortuous. The abdomen was slightly distended and occasional peristaltic waves could be felt. The liver was felt two fingerbreadths below the right costal margin in the midclavicular line. Upon deep inspiration the patient complained of pain across the upper abdomen. There was no subcostal tenderness on either side.

The temperature was 100.8°, the pulse 120. The respirations were 25.

Examination of the urine showed a specific gravity of 1.015 and a very slight trace of albumin. The sediment showed 15 white blood cells and an occasional red blood cell, as well as numerous bacteria. Examination of the blood showed a red cell count of 3,850,000, with a hemoglobin of 65 per cent. There was a white cell count of 30,200, 90 per cent polymorphonuclears. The non-protein nitrogen of the blood was 87 milligrams. The blood sugar was 130 milligrams.

On the day following admission the patient was much worse. The blood pressure had dropped to 80 systolic, and the diastolic could not be obtained. He was given a large amount of five per cent glucose intravenously. A urological consultant did not believe that the prostate was enlarged. There was, however, a residual of about three ounces of urine which was believed to be due to a slightly enlarged median lobe. A questionable tender mass, possibly an enlarged kidney, was felt in the right flank. On the third day the patient became very drowsy and could not be roused. His temperature remained elevated at about 100.5° and his respirations had increased to about 40. He rapidly failed and died on the third day after admission.

DIFFERENTIAL DIAGNOSIS

DR. HOWARD B. SPRAGUE: I should like very much to know what the clinicians who had the privilege of seeing this case thought was the cause of death because I am sure that I do not know.

If we boil this story down, if we can boil down such a meager story, we have a thin, disoriented old man who is dehydrated, anemic and complaining of vague abdominal distress for six weeks, with slight fever and weakness. He comes into the hospital, no adequate history can be obtained, and he apparently does not have whatever benefit he might have received from further study. He dies in three days.

I should think the diagnosis would have to be made on a statistical basis rather than on what one found on examination. Of course at this age one thinks of malignancy and arteriosclerosis, cardiovascular disease, and in this case there is a suggestion of an additional septic factor.

As for the malignancy, we have the age, the marked loss of weight, the abdominal distress, the question of a mass in the right flank and visible and palpable peristalsis. This makes one think of malignancy of the large bowel, although the question of kidney was raised by the consultant.

The arteriosclerosis I think is about the only thing that we can be sure of. We have it in the fundi, in the radials, with the suggestion that it is in the cerebral circulation from the mental attitude, and we have an increased nonprotein nitrogen which is suggestive of arteriosclerotic kidney and a uremic death.

As for sepsis, there is some fever, some pain across the upper abdomen, a markedly increased white count and increased respirations. The degree of dehydration here may account for certain changes in the blood picture, which may be dependent upon the concentration of the blood. I should think that he might very well have had a terminal bronchopneumonia. There is little that we can use to convict the kidney as the source of infection. One perhaps should always think of a cryptic pancreatic episode, which on a vascular basis can sometimes be very confusing, and possibly even of mesenteric thrombosis or embolism. The history seems to be very long for this, however.

As to the heart, the patient did not die a

cardiac death, if by that we mean that he died with orthopnea, or pain in the chest, or with an abrupt exit. We must grant, I think, that the heart was enlarged, the apex being in the axillary line, and there is a murmur which the cardiologist of course seizes upon. It is described as a loud high-pitched systolic murmur heard best at the apex and aortic areas and poorly transmitted to the axilla. We can only say by indirection that it might have been better transmitted to the neck or aortic area. There is a little suggestion that the loudness was more marked at the base. The other point is that there was a regular rhythm and that the beat was forceful. The blood pressure is distinctly noncommittal, I should say. There is a drop to 80 systolic, but every patient who dies has a blood pressure drop to 80 systolic before he dies, presumably. This murmur might be caused by one of various things. One wonders if the anemia does not result in a hemic murmur or whether the patient had a previous hypertension with dilatation of the heart and mitral regurgitation, or whether there is an arteriosclerotic process in the aortic valve or even a left ventricular dilatation from a previous coronary occlusion. However, dilatation seems to me unlikely because of the forceful beat. In dilatation the apex beat is more diffuse usually and not forceful, so that perhaps aortic stenosis is a guess which should be considered, though we know nothing about the thrill, about the heart sounds, about the presence or absence of the aortic second sound, or the character of the pulse. We have had some old people enter this hospital with systolic murmurs who had a rather high degree of calcification of the pericardium which was unsuspected. There is nothing here to indicate that. I will have to say something so I will guess that he might have had abdominal malignancy, generalized arteriosclerosis, uremia, terminal pneumonia and possibly aortic stenosis.

Dr. Tracy B. Mallory: Any case that comes in in a condition where a history is unobtainable puts us off to a very bad start at once. Perhaps Dr. Wheeler can add a little information that we were not able to get out of the records.

Dr. Roy R. Wheeler: I can give little further history. The patient was an old man who lived alone. His son stated he felt that his father had been sick for some time, but nothing very definite was known about it. My impression at the time was that he had an arteriosclerotic nephritis with uremia. I felt that the right upper quadrant condition was an inflammatory process. Physically he showed moderate distention of the abdomen and diffuse pain with some spasm all through the right upper quadrant and flank, with dullness to flatness in the right lower chest. He was too sick for any elaborate investigative procedures and clinically he went steadily downhill and died.

Dr. Channing S. Swan: I saw this patient the day after admission. He was semiconscious. He had not voided for about twelve hours. The non-protein nitrogen was 87 milligrams and it was very suggestive that it might be a case of prostatic obstruction. The prostate by rectal examination, however, did not seem to be enlarged. I catheterized the patient and found a few ounces of urine which could not be said to be residual urine, as the patient did not void for several hours. There was a definite mass in the right flank and upper quadrant which was consistent with the right kidney. I could not make out whether it was tender because the patient was comatose. I suggested that he be given supportive treatment and further examination if he improved. I could not make any diagnosis at that time. There was no positive evidence that he had an enlarged prostate, but without cystoscopic examination it could not be ruled out.

Clinical Diagnoses

Chronic nephritis.
Uremia.

Dr. Howard B. Sprague's Diagnoses

Generalized arteriosclerosis.
Uremia.
Bronchopneumonia.
Abdominal malignancy, (? large intestine).
Aortic stenosis.

Anatomic Diagnoses

Ruptured dissecting aneurysm of the abdominal aorta.
Hematoma of the right diaphragm.
Hemothorax, right.
Arteriosclerosis, generalized.
Aortic stenosis, arteriosclerotic type.
Hypertrophy of the heart.
Pulmonary atelectasis.
Bronchopneumonia.

Pathologic Discussion

Dr. Mallory: I think this patient's condition was entirely undiagnosable on the evidence presented. Even at autopsy it was some time before it was evident what had happened. The right thoracic cavity was full of blood and we could find no explanation for it in any lesion of the lungs, the heart or the thoracic aorta. As we looked at the upper surface of the right diaphragm we found a tumor mass perhaps five inches thick which was soft and deep red in color. On cutting into it the diaphragm seemed to have been separated into two layers by a mass of blood clot. As we traced this blood downward it evidently passed along the right crus of the diaphragm to a point in

apposition with the abdominal aorta at the level of the renal artery, where a rupture of the aorta was discovered.

A PHYSICIAN: You mean that was an aneurysm?

DR. MALLORY: Yes, a dissecting aneurysm limited to a short segment of the abdominal aorta. The rupture of external layers of the aortic wall which is the immediate cause of death in most cases was in this instance not immediately fatal, since the dissection continued up through the diaphragm, and only with the final rupture into the pleural cavity did the hemorrhage become of fatal proportions.

A PHYSICIAN: How long before he actually died do you think that started?

DR. MALLORY: You can almost always separate these histories into two episodes. I think the onset of pain three or four days before death represents the initial period of dissection. Exactly when the outer coat ruptured I cannot be sure, but the perforation into the pleural cavity was probably terminal.

A HOUSE OFFICER: A point which I failed to write into the record concerns his back pain. Every once in a while he would sit up in bed and put his hands across the upper abdomen, even though he was semicomatose, and say, "Oh, my back." We did not know where it hurt, but he did have pain in his back.

DR. MALLORY: That might well have been of real assistance to Dr. Sprague, since back pain is very frequent in dissecting aneurysms.

As to the rest of the autopsy, Dr. Sprague was correct in prophesying severe general arteriosclerosis, hypertrophy of the heart and a moderate grade of senile aortic stenosis. The right lung showed extensive collapse and a moderate amount of terminal bronchopneumonia. A small area of infarction was discovered in the brain in the right lenticular nucleus.

CASE 21012

PRESENTATION OF CASE

A sixty-one year old American housemaid entered complaining of pain in the back of one and a half week's duration.

Ten days before entry the patient fell down a flight of stairs and developed pain in the back. She was not unconscious but was unable to get up although she could move her legs. A physician strapped her back. Her bowels had not moved for about one week. The pain in her back continued and she entered the hospital.

The family and marital histories are non-contributory.

She had the usual childhood diseases including scarlet fever. There was no history of typhoid fever. During the past four years she had developed slight shortness of breath upon exertion. Her appetite had always been poor and she required cathartics to keep her bowels open. She had nausea and vomiting about once a week for the past five months, also attacks of "grabbing" abdominal pain for about one year. Her catamenial history was negative. During the past year she had shooting pains down her legs as well as occasional fleeting pains in her joints. She occasionally had attacks of staggering at which time everything appeared black and spotted before her eyes. The last attack occurred two weeks before entry. For one year she had nocturia two or three times and also vague aches and pains in the extremities. She had lost forty-three pounds during the past three years.

Physical examination showed a well-developed and fairly well-nourished woman in no distress. The mucous membranes were slightly pale. The lungs were clear except for a few moist râles at the bases. The heart was not enlarged. There was a loud, blowing systolic murmur at the apex, but it could also be heard all over the precordium. No diastolic murmur could be heard. The apex beat was quite diffuse and forceful. There was moderate tenderness over the lower ribs on the right side. Pelvic examination showed moderate tenderness in the right vault and a vague sense of resistance. There was a profuse yellowish, thick vaginal discharge.

The temperature was 98.6°, the pulse 88. The respirations were 20.

Examination of the urine showed a specific gravity of 1.010 to 1.012 with a large trace of albumin. A sediment showed 6 to 8 white blood cells and an occasional cast. The blood showed a red cell count of 2,140,000, with a hemoglobin of 55 per cent. There was a white cell count of 18,800. A smear showed marked achromia and slight anisocytosis of the red blood cells. The platelets were slightly increased. The white blood cell differential showed 62 per cent polymorphonuclears, 25 per cent lymphocytes and 13 per cent blast forms. A stool examination was negative. A Hinton test was negative. The serum protein of the blood was 11.7 per cent.

X-ray examination of the chest showed slight cardiac enlargement. There were no mediastinal masses. The lung fields were clear. A gastrointestinal series was negative. There was rather wide trabeculation in the sacrum but no increase in the size of the bone or any evidence of metastases. Films of the shoulder girdles showed generalized atrophy and several small punctate areas of diminished density. A lateral view of the skull showed numerous rounded, sharply defined areas of diminished density together with some atrophy.

Further blood smears showed plasma cells with occasional normoblasts. On the sixth day Bence-Jones protein was found in the urine. The non-

protein nitrogen of the blood was 107 milligrams per cent. She rapidly failed, became comatose, developed signs of bilateral bronchopneumonia, and died on the tenth day.

Differential Diagnosis

Dr. William D. Smith: A sixty-one year old housemaid enters complaining of back pain precipitated by a fall ten days previously. We are not told whether her pain was over the sacrum or between the shoulder blades. If pain in the back were worth recording at all at least we should be told the location of the pain. I should be inclined to ignore this complaint were it not for the fact that on reading on one suspects a condition in which fracture might easily occur with relatively slight trauma. The fact that she could move her legs suggests that there was no severe cord damage.

During the past four years she has developed slight shortness of breath on exertion. This raises the question of heart, lungs, anemia, or general weakness.

There had been slight gastro-intestinal symptoms as suggested by occasional nausea and vomiting and attacks of "grabbing" abdominal pain for about a year. This statement is entirely too vague to permit us to speculate on the cause of these symptoms but I would make the same comment on the "grabbing" abdominal pain that I did on the back pain, that is, if this pain was regarded as of significance, certainly its location, severity, duration and possible radiation should have been recorded.

The story of shooting pains in the legs and fleeting joint pains of the past year is too vague to be helpful.

The occasional staggering, when things appeared black and spotted before her eyes, raises the question of real vertigo, anemia, general weakness or perhaps sudden hemorrhage.

If she was a poor sleeper or kept awake by aches and pains, her nocturia may mean nothing. If, on the other hand, she was awakened two or three times at night by the necessity of voiding, we might assume urinary infection or nephritis.

The loss of forty-two pounds makes one think of malignancy, although it is not unusual for ageing people developing arteriosclerosis to lose a good deal of weight.

The physical findings show pallor of the mucous membranes, a few moist râles at the lung bases, with a loud, blowing systolic murmur audible all over the precordia. Rheumatic heart disease would be unusual in a woman of sixty-one and this systolic murmur suggests arteriosclerotic heart disease or perhaps anemia or possibly both combined. The presence of moist râles at the lung bases suggests a slight degree of congestive failure, particularly when considered in relation to her increasing shortness of breath on exertion.

Tenderness over the lower ribs on the right may be the result of trauma from her fall or, as we read farther in the history, may suggest the possibility of pathology in the ribs themselves.

Pelvic examination I will not comment upon other than to say that there is nothing in the vaginal discharge to suggest carcinoma. It was not foul or bloody. I suppose that she may have had an endocervicitis, possibly a pelvic infection or tumor, perhaps even an infection with trichomonas vaginalis although here again there is nothing characteristic of this in the description of the discharge.

We note normal respiration, slightly accelerated (88) pulse, and normal temperature.

The urine analysis with its large trace of albumin suggests nephritis but one is struck by the rather striking absence of formed elements in the microscopic sediment as compared with the large amount of albumin. This feature does not, of course, rule out a chronic nephritis.

The blood examination shows a severe grade of anemia with a hemoglobin of 55 per cent, a red cell count just over two million, and a white cell count of 18,800, with platelets slightly increased and marked achromia. (This illustrates what we so often see in our blood reports. As indicated by the red count and hemoglobin the patient has a high color index and yet the examination of the smear reports marked achromia.) I am going to bank on the smear and assume that she has an anemia definitely of the secondary type. The differential count is not remarkable except for the presence of 13 per cent of blast forms. In any profound anemia an occasional immature white cell may be present but 13 per cent of immature cells deserves respect. A white cell count of 18,000 in the absence of fever suggests that the leukocytosis is due to something other than an infectious process. Taking the blood picture as a whole, pernicious anemia is obviously out of the question. The increased white cell count and the increased platelets might suggest acute blood loss but again we have 13 per cent of blast forms which points to some bone marrow disturbance. Such a picture is compatible with a leukemia with an unusually low white count, or malignancy with bone marrow metastases, or perhaps with myeloma.

The serum protein at 11.7 per cent, if correct, is almost double the normal. I do not know very much about such an increase but it certainly indicates a profoundly altered metabolism and perhaps may be explained by a greatly increased endogenous protein katabolism. Abnormally high serum protein has been reported in some cases of myeloma.

There is nothing in the x-ray examination of the chest that need detain us. The trabeculation in the sacrum I should regard as the changes of increasing age. The skull plates

showing numerous rounded, sharply-defined areas of diminished density make one think of possible myeloma or of metastatic carcinoma from some unknown primary focus. I have a notion, however, that the sharply defined character of these areas is rather more suggestive of the myeloma tumor than of metastatic carcinoma, which I think would tend to have more irregular or ragged outlines.

I note that further blood smears identified these blast forms as plasma cells with occasional normoblasts, again suggesting bone marrow disturbance. Furthermore, if one regards multiple myeloma as a plasma cell tumor, one might perhaps regard them as tumor cells in the peripheral blood.

The appearance of Bence-Jones protein in the urine makes us immediately consider the classic condition in which this appears, that is, multiple myeloma. On the other hand, we must not forget that it may occur in carcinomatosis with metastasis to the bone marrow, in chloroma, in osteosarcoma originating in the bone marrow, and occasionally in leukemia.

A non-protein nitrogen of 107 milligrams per cent in connection with the large trace of albumin again makes us think of the possibility of nephritis, but an increased nitrogen may occur in leukemia or possibly in any condition in which the endogenous protein katabolism is speeded up. Incidentally, the severe nephritis sometimes associated with myeloma must be kept in mind.

To sum up, we have a woman of sixty-one obviously going downhill for the past two or three years with weight loss, vague pains in her limbs, a little shortness of breath, a severe secondary anemia with 13 per cent of immature cells later recorded as plasma cells, high serum protein and high non-protein nitrogen, Bence-Jones proteinuria, and an x-ray consistent with multiple myeloma which seems an almost obvious diagnosis. However, when we talk about myeloma we are pretty close to the leukemic group and myeloma has been reported with a blood picture of a plasma cell leukemia; so perhaps Dr. Mallory may show us some unusual type of leukemia associated with bone tumor. Further, one has to think of the possibility of carcinoma with metastasis to bone and bone marrow associated with Bence-Jones protein. It seems to me that there is nothing in the record to suggest other causes of bone rarefaction such as osteomalacia, osteitis fibrosa cystica, or hyperparathyroidism.

Clinical Diagnoses

Chronic nephritis with uremia.
Multiple myeloma.

Dr. William D. Smith's Diagnoses

Multiple myeloma.
General arteriosclerosis.
? chronic nephritis.
? pelvic pathology.
? terminal bronchopneumonia.

Anatomic Diagnoses

Multiple myeloma.
Myeloma of kidney.
Myelomatous infiltration of spleen.
Pulmonary infarct.
Arteriosclerosis, aortic, slight.

Pathologic Discussion

Dr. Tracy B. Mallory: Multiple myeloma is a disease in which the history is rarely of much diagnostic value; whereas there are numerous strongly suggestive or even diagnostic laboratory procedures. The work of Magnus-Levy, Bell and others has called attention to the very frequent association of a specific type of renal pathology in association with myeloma. So far as I know it is limited to the myeloma cases which show Bence-Jones protein in the urine. These cases show a mechanical plugging of the collective tubules with large hyalin casts of exceptional density. The tubules above the point of blocking are apt to be distinctly dilated, and this may be so marked that the condition has occasionally been described as intranephric hydronephrosis. This patient showed such a condition although in not so marked a degree as some other cases we have seen. Another point of considerable interest in the case was the appearance of plasma cells in the circulating blood. I believe it would have been possible to make a flat-footed diagnosis of myeloma from an examination of the blood smear alone. As is often the case, however, these plasma cells were not recognized by several examiners until Dr. Hunter pointed them out. In our own examination of the blood we found about five per cent of plasma cells, rare myelocytes and quite numerous nucleated reds which were not reported in the hospital differential count. Occasionally the invasion of the blood stream with plasma cells may be so great that a diagnosis of plasma cell leukemia is justified. This type of blood-stream invasion is most apt to occur in the cases where the myelomatous infiltration of the bone marrow is diffuse without the formation of the multiple focal tumors which can be recognized by x-ray examination. In this case the bone marrow showed both localized tumor formation and diffuse infiltration. Infiltration may appear in the internal organs but is not usually extensive. In this patient many atypical cells, only a few of which could be positively recognized as plasma cells, were found in the spleen while the liver and lymph nodes were uninvolved. The only other finding of significance was a small pulmonary infarct.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

COMMITTEE ON PUBLICATIONS

R. I. LEE, M.D., *Chairman* HOMER GAGE, M.D.
R. B. OSGOOD, M.D. R. M. SMITH, M.D.
F. H. LAHEY, M.D.

EDITORIAL STAFF

REID HUNT, M.D. STEPHEN RUSHMORE, M.D.
JOHN P. SUTHERLAND, M.D. HANS ZINSSER, M.D.
GEORGE R. MINOT, M.D. BENJAMIN WHITE, PH.D.
FRANK H. LAHEY, M.D. HENRY R. VIETS, M.D.
SHIELDS WARREN, M.D. ROBERT N. NYE, M.D.
GEORGE L. TOBEY, JR., M.D. ROBERT M. GREEN, M.D.
C. GUY LANE, M.D. CHARLES C. LUND, M.D.
WILLIAM A. ROGERS, M.D.
WALTER P. BOWERS, M.D., *Managing Editor*

ASSOCIATE EDITORS

GEORGE G. SMITH, M.D. WILLIAM B. BREED, M.D.
JOSEPH GARLAND, M.D.

THE NEW HAMPSHIRE MEDICAL SOCIETY
PUBLICATION COMMITTEE

CARLETON R. METCALF, M.D. HENRY H. AMSDEN, M.D.
WARREN H. BUTTERFIELD, M.D.

THE VERMONT STATE MEDICAL SOCIETY
PUBLICATION COMMITTEE

WILLIAM G. RICKER, M.D C. F. DALTON, M.D.
L. H. ROSS, M.D.

SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

WILLIAM HARVEY'S CHURCH

WILLIAM Harvey died June 3, 1657 and was buried in Hempstead Church in Essex. The Fellows of the College of Physicians of London followed his body to the church, where it was wrapped in lead and laid in the family vault. There it remained until October 18, 1883, when several Fellows of the College, in the presence of the President, Sir William Jenner, saw the body transferred to a white marble sarcophagus and placed in a special chapel erected by the College. The church has become a shrine to physicians from all parts of the world. Here lies one of the greatest of all men of medicine and everything connected with the building has a special appeal to the medical world.

The church was originally constructed in the fourteenth century. Its ancient tower collapsed in 1882. About ten years ago a movement was set on foot to rebuild this tower and some local subscriptions were collected to that end. At the time of the Harvey tercentenary in 1928, celebrating the publication of "De Motû Cordis", an additional impetus was given to this movement by the Royal College of Physicians of London. As soon as additional donations were received, the work of reconstruction was begun and the tower is now partly finished and only awaits further funds to complete it. As noted in the *Lancet*, November 3, 1934: "The present appearance of the structure seems to convey a reproach to us all from the great dead. Accordingly a further appeal for the sum of $10,000 necessary to finish the work is made to the whole medical profession. The members of the Harveian Society of London, to mark their centenary in 1931, instituted a special fund for the reconditioning and rehanging of the bells." It is hoped that this appeal will be widely noticed in America.

Proposed restoration of tower of Hempstead Church, Essex, England. The black line indicates the approximate height to which the tower has now been built*.

Donations are invited from all members of the medical profession and should be made payable to the Harvey Memorial Fund and sent to Dr. G. de Bec Turtle, Royal College of Physicians of London, Pall Mall East, London, S. W. 1.

*Courtesy of *The Lancet*.

QUESTIONABLE INSURANCE POLICIES

IN the November 22 issue of this *Journal*, page 983, attention was called to the selling of insurance policies in Massachusetts by a company not licensed in this state.

Current reports in the daily press indicate that a large number of people in Bristol County have bought policies issued by a company which has repudiated the claims of the holders.

State Commissioner of Insurance, Merton L. Brown, has explained that there is difficulty in suing a company of this character, for action

must be taken in the state where the company is chartered, and again the warning is issued that residents of Massachusetts should not purchase insurance from a company which is not licensed to transact business in this state. Records are on file in the State Department of Insurance respecting licensed and unlicensed insurance companies. Policies in unlicensed companies are not to be depended on. There are too many ways of losing money to take a chance with insurance companies which have no standing in the state where the policies are sold. The old adage that "the buyer must beware" is especially appropriate in the insurance field.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WEBBER, ISAAC M. B.S., M.S., M.D. Bowdoin Medical School 1920. F.A.C.S. Surgeon, Maine General Hospital, St. Barnabas Hospital and Children's Hospital. Consulting Surgeon, Webber Hospital, Biddeford, Maine. His subject is "The Limitations of Enterostomy and Undesirable Effects Incident to Its Use." Page 1. Address: Leighton Building, Portland, Maine.

STURGIS, SOMERS H. M.D. Harvard University Medical School 1931. Member of Staff, Out-Patient Department, Cambridge Hospital. Address: 3 Pinckney Street, Boston, Massachusetts. Associated with him is

LUND, CHARLES C. A.B., M.D. Harvard University Medical School 1920. F.A.C.S. Surgeon, Collis P. Huntington Memorial Hospital. Assistant Visiting Surgeon, Boston City Hospital. Instructor in Surgery, Harvard Medical School. Address: 319 Longwood Avenue, Boston, Massachusetts. Their subject is "Leukoplakia Buccalis and Cancer." Page 7.

FREEDMAN, HAROLD J. B.S., M.D. Harvard University Medical School 1927. Formerly, Assistant Pediatrician at the Boston Dispensary, and Pediatrician in the Out-Patient Department of the Beth Israel Hospital. Now Assistant Physician, Medical Department, Children's Hospital. His subject is "Acute Anaphylactic Shock Following Intracutaneous Test for Sensitivity to Horse Serum. Report of a Fatal Case." Page 10. Address: 371 Commonwealth Avenue, Boston, Massachusetts.

WHITE, CHARLES E. M.D. Boston University School of Medicine 1929. His subject is "The Relation of Arcus Senilis to Arteriosclerosis and Senility." Page 10. Address: Taunton State Hospital, Taunton, Massachusetts.

LOMAN, JULIUS. M.D. Tufts College Medical School 1925. Instructor in Neurology, Tufts College Medical School. Psychiatrist, Research Staff of Boston State Hospital. Assistant in Neurology, Beth Israel Hospital. His subject is "Progress in Neurology in 1933." Page 13. Address: 270 Commonwealth Avenue, Boston, Massachusetts.

MISCELLANY

PROCEDURE FOR CONTROLLING BOVINE MASTITIS

Owners of cattle affected with mastitis, a disease of the milk glands, may take advantage of Federal emergency funds for combating the disease through the assistance of the U. S. Department of Agriculture. This work is authorized by a new regulation designated as B. A. I. Order 351, to be effective January 2, 1935. The order, which will be administered by the Bureau of Animal Industry and coöperating State and local sanitary officials, provides that any owner may apply for the examination of his herd by an accredited veterinarian representing the Bureau or State. Cows that show marked physical evidence of mastitis will then be removed and slaughtered and indemnities paid to the owner according to the appraised value.

Funds amounting to $800,000 for conducting this work and for the payment of indemnities have been allotted by the Agricultural Adjustment Administration out of money made available by the La Follette amendment to the Jones-Connally Act. Similar emergency work was begun during the summer with bovine tuberculosis and Bang's disease of cattle, and the new order will extend the disease-control work to another cattle disease that is the source of heavy losses to dairymen.

CORRESPONDENCE

POOR RELIEF AND MEDICAL CARE

December 26, 1934.

Editor, *New England Journal of Medicine*,

The letter of Dr. Pavlo in this week's *Journal* is quite timely and pertinent to conditions as existing in Boston.

There was an offer to the Mayor by representatives of most of the physicians in the Boston Area to treat the indigent poor of Boston at the nominal rate of $1 per visit, the same to be paid by the City.

It was ruled by the City Law Department, that the City could not under the Charter spend any money for physicians to treat the indigent of the City.

The Shadman Committee then, on behalf of the aforementioned group of physicians, offered to take care of the poor of Boston, without cost, providing the City Hospital would exclude all but the indigent poor and injured, according to the Charter of the Boston City Hospital.

This in my opinion would be the best solution of the difficulty, as we hope there is a limit to this de-

pression and that these unfortunates will not always be on the Welfare and the fact that most of the Welfare cases are now treated free by physicians.

However, Dr. Wilinsky, as Dr. Pavlo says, approved of the City Hospital taking care of the poor, and this plan had the complete endorsement of the Boston Health League — that great friend of the physicians of Boston!

I can visualize what would happen if this plan had gone through. We just have to observe the workings of Dr. Wilinsky's Baby Hygiene. Do we general practitioners have any baby carriages stop at our doors during office hours now? Do people come to have their children vaccinated? Do we have any toxin-antitoxin work to do? No! Dr. Wilinsky and his Corps of Nurses have attended to that and we not only lose this work but have to pay taxes to support the racket.

Notwithstanding your Editorial note, Dr. Pavlo is entirely right, one well-paid job is enough to hold at any time. How much more true is this now when so many of our colleagues are in financial difficulties!

CHARLES MALONE, M.D.

46 St. John Street,
Jamaica Plain, Mass.

A COMMENT ON CABOT CASE HISTORIES

New York,
December 10, 1934.

Editor, *New England Journal of Medicine*,

Just a couple of critical remarks after reading the last Cabot case histories. (1) Cancer increases with age. The proportion of deaths from cancer to living in the various age groups increases steadily from birth to death. Vide: Ewing: relation of old age to cancer: Am. J. Med. Sc., within a year or two. Also, in pure Boston why do they call cancer "malignancy"? Malignancy is an attribute, an abstraction. Bubonic plague is malignant and so are many cancers. I am profoundly shocked.

Sincerely yours,
F. S.

THE TRANSLATION OF FRACASTORO'S SYPHILIDIS

December 27, 1934.

Editor, *New England Journal of Medicine*,

In a recent book review of "The Sinister Shepherd" a translation of Girolamo Fracastoro's Syphilidis Sive De Morbo Gallico Libri Tres, by William Van Wyck, printed in the *New England Journal of Medicine*, page 651, issue of October 4, 1934, the statement is made that "there are still other and equally good translations available, although not in English". I wish to call attention to the translation made by Dr. Wilmer Cave Wright "Fracastoro, Girolamo 1483-1553. De contagione et contagiosis morbis et eorum curatione, libre III; translation and notes by Wilmer Cave Wright. (Hist. of medicine ser., No. 2) 1930, Putnam"; and to that of Dr. Martin "Syphilis; a translation in prose from the original Latin of (his) immortal poem; with a history of (his) life by Mario Truffi, etc. Translated by S. C. Martin. Second edition revised and enlarged 1931, Urologic and Cutaneous Press, St. Louis."

Sincerely yours,

MADELAINE R. BROWN, M.D.

412 Beacon Street,
Boston, Mass.

EDITORIAL NOTE: The above letter has been called to the attention of the reviewer. It is particularly regrettable that these references were not given in the original review and the reviewer can only plead carelessness in not referring to them. It might be pointed out that the translations into other languages are, on the whole, much superior to those into English.

THE WRITER OF THE REVIEW.

LIAISON OF MEDICINE AND THE PRESS

Time
The Weekly Newsmagazine
New York-Chicago
Editorial Offices
135 East 42nd Street
New York City

December 22, 1934.

Editor, *New England Journal of Medicine*,

The kind appreciation of *Time's* reporting of Medicine's news which you made in the November 15 issue of the *New England Journal of Medicine* came to my attention last month. My first impulse, which I suppressed for fear of intruding among sensitive committeemen, was to applaud your recommendation of a publicity committee for the Massachusetts Medical Society and to offer what information and advice I have regarding such a committee's utility and function.

Such liaison between Medicine and Press should exist in every community, especially now when social medicine is being discussed. The liaison should be constant and alert, and should be stimulated as often by the doctors as by the editors.

The set-up you suggest resembles the New York Academy of Medicine's committee on public information. Your investigators doubtless will go to Manhattan to study the local mechanism. When they are here I shall gladly give them my viewpoint. Or, if you prefer, I shall gladly go to Boston to address them. If the latter, a Tuesday or a Wednesday would be best for me.

With best salutations of the season,

Sincerely yours,

MYRON WEISS, *Associate Editor.*

"LIVER THERAPY IN ANEMIA"

Editor, *New England Journal of Medicine,*

Dr. Trumper* fails to give the exact reference in the *Talmud* wherein it states "For anemia have patients eat the viscera of creatures."

The word "*jârâq*" or "*jêrâqôn*" or jarqônâ may refer to "green" or anemic pallor, or *anemia.*

In Joma 84a Rabbi Mathja Ben Cheresch advised the use of *liver* (and meat) of an ass for one bitten by a mad dog. The "wise men" did not agree with this treatment.

In Sabbath 134a, Rabbi Nathan (Habavli, — of Buvel) speaks of *jârâq,* (*anemia*) in a hemophilic newborn male infant, and advises against circumcision.

Very truly yours,
HYMAN I. GOLDSTEIN, M.D.

Camden, New Jersey,
December 20, 1934.

*Trumper, A.: New Eng. J. Med. 211:1068 (Dec. 6) 1934.

NOTE: A. Cornelius Celsus, in his work "On *Medicine*" (Book IV, Caput IV)—says "Nor it is groundless opinion that a *fox's liver,* after being dry, and deprived of its juice, ought to be bruised, and the polenta from it sprinkled in the drink . . . "Est etiâ nô uana opinio uulpium iecur ubi siccû aridum factum est; contundi oportere: polentâque ex eo potioni aspgi": . . .

RECENT DEATHS

FAMOUS MEDICAL JOURNAL EDITOR DIES

DR. LEWIS STEPHEN PILCHER

Dr. Lewis Stephen Pilcher, scholar and editor for half a century of the oldest surgical journal in the United States, the *Annals of Surgery,* died December 24, 1934, at eighty-nine years of age. Country school teacher, country practitioner, naval surgeon, student of tropical disease, anatomist, professor of surgery, editor, bibliophile, patriot—these nouns indicate a few of his many interests and activities over a long and intensely useful life.

Lewis Stephen Pilcher entered the University of Michigan at the age of thirteen, and took his bachelor's degree at seventeen, the youngest matriculant and the youngest graduate of that institution. His master's degree was added within a year; and in the same year he entered upon medical study. This was in 1863 when the Civil War was raging. The next year found him a volunteer hospital steward. Then back to the University of Michigan and the doctor's degree in 1866. In 1900, this same institution conferred upon him the honorary degree of Doctor of Laws. Practice began in a rural district of Michigan at the age of twenty; at the same time, to guarantee a livelihood, teaching in the little schoolhouse by the blacksmith shop. He rode his horse across the countryside to the call of the sick, followed the current literature of medicine, and for diversion read the classics in their original Greek and Latin.

The next move was to an interneship in a Detroit hospital. Then a postgraduate course in the hospitals of New York City. And then came the successful examination and appointment as Assistant Surgeon in the United States Navy, in 1867. His retirement from the Navy and entrance into private practice in 1872 followed. He organized a dissecting room in his house. This expanded into an adjacent building. A museum and library grew up in connection with it. He dissected also at the Long Island College, and became Adjunct Professor of Anatomy, in 1879, and Surgeon to the Dispensary. In 1885 he was appointed Professor of Surgery at the New York Post Graduate Medical School.

In 1884 he became editor of the *Annals of Surgery,* which position he occupied to the time of his death. This publication, beginning in 1884, was acquired in 1897, by J. B. Lippincott Company. The editorial policy and censorship of advertising have never been relinquished by the editor. If we add to the fifty years of the *Annals of Surgery,* the seven years of the *Annals of Anatomy and Surgery,* and its predecessor which he inspired and dominated, this period of medical editorship establishes him as the dean of medical editors in the United States, if not in the world.

McGINITY—JOSEPH TANEY MCGINITY, M.D., of 146 Chestnut Street, Springfield, Massachusetts, died December 24, 1934. He was born in 1880 and graduated from the University of Vermont College of Medicine in 1905. He joined the Massachusetts Medical Society in 1914; was also a member of the Vermont State Medical Society, and a Fellow of the American Medical Association. He had been a member of the Staff of Mercy Hospital. He was a member of the Springfield Lodge of Elks, Archbishop Williams Assembly, the Knights of Columbus, and the Calvert Club.

He is survived by his widow, Mrs. Anna Leary McGinity; three sisters, Mrs. Mary Leonard of Mechanicsville, N. Y.; Miss Katherine McGinity of Thompsonville, Mrs. John Hughes of Pittsfield, and a brother, Patrick, of Thompsonville.

WINSLOW — EDWARD SMITH WINSLOW, M.D., of Harwichport, Mass., died December 28, 1934. He was born in Easthampton in 1866, the son of Dr. Joseph Winslow and Mrs. Emily Winslow, and graduated in medicine from the Dartmouth Medical School in 1892 and later studied at the Women's Hospital of London, England.

He served with the Army Medical Corps in the World War and was a past commander of the Harwich Post, American Legion. He also served on the staff of the Cape Cod Hospital in Hyannis.

He joined the Massachusetts Medical Society in 1893. He was a member of the Easthampton Masonic Lodge and the Hyannis Lodge of Elks.

His religious affiliations were with the Harwichport Pilgrim Church.

Dr. Winslow is survived by his widow, Mrs. Edith Watson Winslow; a son, Edward L. Winslow, and a sister, Mrs. Edmund Sawyer.

NOTICES

IMPORTANT NOTICE

See Erratum on page ix, advertising section.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, January 10, in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

LECTURES AND CLINICS ON HEART DISEASE BY DR. CHRISTIAN

Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic at the Harvard Medical School, will give a series of 13 lectures and clinics on heart disease at the Peter Bent Brigham Hospital as follows: January 14 at 8:30 A.M., January 17 at 4:30 P.M., January 21 at 8:30 A.M., January 24 at 4:30 P.M., January 31 at 4:30 P.M.; February 7 at 8:30 A.M. and 4:30 P.M., February 14 at 8:30 A.M. and 4:30 P.M., February 18 at 8:30 A.M., February 21 at 8:30 A.M. and 4:30 P.M., and February 25 at 8:30 A.M. Physicians are cordially invited.

The subjects discussed will be the following:

1. Physiology of heart in relation to normal and abnormal cardiac function.
2. Symptoms and signs of cardiac disease; varieties of heart disease and their diagnosis.
3. Treatment of cardiac insufficiency with discussion of pharmacological action of chief therapeutic agents used.
4. Special forms of heart disease; angina pectoris and coronary occlusion.
5. Special forms of heart disease (continued); chronic non-valvular heart disease including effects on heart of arteriosclerosis and of hypertension.
6. Special forms of heart disease (continued); chronic valvular heart disease; syphilitic heart disease.
7. Special forms of heart disease (continued); acute endocarditis; acute and chronic pericarditis.

REMOVAL

DWIGHT O'HARA, M.D., announces the removal of of his office from 520 Commonwealth Avenue, Boston, to 5 Bay State Road, Boston.

REPORTS AND NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society was held in the amphitheatre of the Peter Bent Brigham Hospital on the evening of December 11, 1934. Dr. Channing Frothingham called the meeting to order. The first case to be presented was that of a fifty-two year old female who had had diabetes for the past seven years with dietary treatment during this period, and insulin for seven weeks. On entry she had lost about a hundred pounds, was easily fatigued, and had had a sudden hemoptysis three weeks before entry. Physical examination disclosed an emaciated woman showing no abnormal clinical pulmonary signs, but a positive x-ray and sputum that was positive for tubercle bacilli. Dr. M. C. Sosman said that this patient represented the "unsensitized" type of tuberculosis which is seen in children and in some diabetic adults.

The second case was that of a twenty-seven year old married female, who had had an attack of pneumonia five years ago, and a thickened pleura by x-ray two years ago. After a prolonged cold last winter, her cough became productive of a sputum positive for tubercle bacilli, and by x-ray she had signs of tuberculosis. Pneumothorax followed by temporary phrenic paralysis had been carried out, and she entered the hospital at this time for a thoracoplasty. There were signs of cavity on the left side. The first and second stages of a thoracoplasty had been carried out with the removal of the first to the eighth ribs. Dr. Newton discussed the necessity of selecting the proper case for thoracoplasty, and the safety of multiple operations. He said that the ribs are resected only over the diseased part of the lung.

Dr. Gerardo M. Balboni spoke on "The Development in the Treatment of Pulmonary Tuberculosis from 1696 to the Present Time." Georgius Baglivi in 1696 was the first to propose treatment by means of a transverse incision between the ribs. Samuel Sharp in 1747 advised incision and drainage of the abscess, there being very little distinction between tuberculosis and pulmonary abscess at this early date. Ebenezer Gilchrist in 1777 suggested a sea voyage as the best means of clearing up pulmonary infection.

It was James Carson who in 1819 insisted on operative surgery in the treatment of this condition, and proposed pneumothorax. He treated two cases successfully in this way, but his work was forgotten until 1909 when it was rediscovered. Many substances were injected by various men at different times in the nineteenth century with uniform failure to procure satisfactory results.

Forlanini in 1894 reported the first successful case of artificial pneumothorax, and he used small amounts of nitrogen at frequent intervals. The beneficial results of spontaneous pneumothorax and hydrothorax had been recognized previous to this time.

Robinson in the early part of the present century used artificial pneumothorax at the Massachusetts General Hospital, and gave larger amounts of nitrogen. Dr. Balboni has also been very active in this work.

Dr. Reginald Fitz then spoke on "The Library May Afford a Certain Amount of Entertainment." He delivered a most fascinating lecture and showed how the library may be a place in which to have real fun. He based his talk on the life of Dr. John H. Watson of Queen Victoria's army, who received his medical education from the London University Medical School, from which he graduated in 1878. After a series of exciting adventures in the Afghanistan War, from which he barely escaped with his life, he returned to London where he met a certain Sherlock Holmes. These two men set up a lasting alliance about which the average American citizen knows a good deal. Dr. Watson introduced effective occupational therapy for neurasthenia on himself by busily taking notes of their numerous adventures. About seven years after their meeting he married a young lady three days after he had met her, and settled down to a practice which never really fully engaged his adventuresome mind. He then began to write up the various escapades of Holmes and himself, and frequently went with Holmes on new adventures. Soon after the supposed killing of Holmes by Moriarty, Watson's wife died, but Holmes reappeared three years later, and they again took up their quarters on Baker Street, where they lived for several years until Watson was married again to a mysterious girl about whom little is known. From this time he gradually broke away from Holmes, and faded away into obscurity.

Dr. Fitz considers Dr. Watson as a real doctor, one of the greatest, and a real personality.

THE DUNHAM LECTURES

Dr. Ulrich Friedemann, formerly Professor at the University of Berlin and Director of the Division of Infectious Diseases, Virchow Krankenhaus, delivered the three Dunham Lectures for 1933-34 at the Harvard Medical School on the afternoons of December 4, 6 and 11.

After a brief introduction by Doctor Zinsser, Doctor Friedemann opened the first of his lectures on "The Distribution of Dyes in the Body". He believes that distribution is essentially regulated by a selective permeability of the vascular system and that the factors controlling this selective permeability are the same for dyes, toxins, drugs, and viruses. He studied in particular the selective vascular permeability in the brain.

Certain toxins are very poisonous to the brain cells if brought into direct contact with them, while having no influence if introduced into the blood stream. The conception that other organs absorb these toxins and thus protect the brain is refuted by the fact that the toxin stays in the blood stream a long time.

There are two barriers between the brain tissue and its surrounding tissues: first, the blood-brain barrier which consists of the vessel walls; and secondly, the spinal fluid barrier. Some toxins are much more toxic intrathecally than intravenously. By injecting certain dyes into the blood stream of animals, Doctor Friedemann found that they stained the brain while the spinal fluid was free from the dye, thus indicating to him that the important route from the blood to the brain is directly through the vessel walls, and he set out to determine the factors controlling the selective permeability of this barrier. By injecting varying doses of several different dyes intravenously, he found that with the exception of aurantia, acid dyes did not tend to be absorbed at all or only when very high doses were used, while practically all of the basic dyes stained with only a very small dose. The affinity of the brain cells for these same dyes was then tested, and it was found that on the whole the basic dyes stain in far greater dilution than the acidic dyes. By a comparison of the doses needed directly with those needed by the intravenous route, the permeability of the vessels to the different dyes was determined, and it was concluded that the blood-brain barrier is more permeable to the basic dyes. The two basic dyes which passed the vessel walls with the greatest difficulty are amphoteric (malachite green and pyronin). It was found that colloidal or non-colloidal character of a dye was of no significance, and the size of the particle had no influence.

Doctor Friedemann investigated the phenomenon of irreciprocal permeability by which certain substances will pass one way through a membrane but not the other. A potential gradient exists within the membrane and basic substances will pass only from the inner to the outer layers of the membrane while acid substances act in a reverse fashion. The blood-brain barrier is only slightly permeable to the antibodies of diphtheria and to the toxin of this disease when going from the blood to the brain but is permeable in the opposite direction. A much higher antibody titre is obtained when immunization is carried out intracerebrally than when it is carried out extracerebrally.

The second lecture of the series considered "The Distribution of Drugs, Toxins, and Viruses in the Animal Body." It was found that the drugs which act on the brain are to a large extent basic and that when acidic radicals are inserted, they are often detoxified. Certain drugs which are not toxic when introduced by the intravenous route become so when injected intrathecally.

The minimum lethal doses of toxins were compared when injected intravenously, intrathecally, and intracerebrally and it was found that toxins were usually five to ten times as toxic by the intrathecal route and twenty to forty times as toxic by the intracerebral route as by the intravenous method of injection. The blood-brain barrier is impermeable to the toxins of diphtheria, tetanus, botulinus, and

meningococcus, but is permeable to the toxins of cobra venom and lamb dysentery.

Insulin and thyroxin had no effect on the permeability of the brain capillaries, while adrenalin and pituitrin caused a marked increase in the permeability to dyes, alcohol, paraldehyde, urethrane, strychnine, cobra toxin, and lamb dysentery toxin. These substances became five to ten times as toxic as they were before the injection of the adrenalin or pituitrin, but these hormones had no effect where the blood-brain barrier was impermeable before the injection.

It was found that most toxins have a negative charge while cobra venom carries a positive charge and is a base. These findings suggested that only those toxins which are basic or which are isoelectric can pass the blood-brain barrier. The electric charge also has some effect on the length of the incubation time. Many of the viruses cannot pass the blood-brain barrier and several reach the brain via the peripheral nerves. Their inability to pass the vessel walls is probably not due to their size but to their charge and certain other unknown factors.

The third lecture was on "The Pathogenesis of Diphtheria." The intravenous injection of from 500 to 1000 minimal lethal doses of diphtheria toxin does not lead to the presence of any toxin within the brain and therefore can have no effect via the brain on the circulatory failure seen in this disease.

Dr. Friedemann studied many cases of malignant diphtheria in Germany and found that there are three stages of the disease in which circulatory failure may occur. The first stage is from the first to the fourth or fifth day and circulatory failure in this stage is very fatal. Except for the occasional finding of traces of cellular infiltration or fatty degeneration, there are no histological or electrocardiographic changes, and heart failure in this type is thought to be due to an insufficient venous return to the heart rather than to the effect of the toxin. In the second stage of the disease, from the fifth to the tenth to fourteenth days, circulatory failure is accompanied by irregularities of the heart beat without cardiac dilatation. The electrocardiogram shows changes in the conductive system with varying types of heart block. Complete heart block may occur without any change in the pulse rate, and can be told only by the electrocardiograph. Histological study shows definite lesions in the conductive system in all cases. These lesions consist of foci of cellular infiltration within the bundle of nervous tissue with very little change in the myocardium, although occasionally there are small areas of infiltration in the muscle also. There are often hemorrhages from the smaller branches of the coronaries. The liver may demonstrate similar hemorrhagic and infiltrative changes. Death from heart failure in this second stage is not due to toxin in the heart muscle but rather to lesions in the coronary vessels.

In the third stage, after the fourteenth day, the electrocardiogram may show sinus arhythmias, a smaller amplitude of the ventricular complex, and an inverted T-wave in the second lead. The histologic changes are extensive with hyalin degeneration of the heart muscle fibres and vascular lesions of the coronary vessels are suggested. There is extensive fibrosis of the heart and it is believed that the heart failure of this stage is not due to toxin but perhaps to the vascular lesions, although it has been commonly thought that the postdiphtheritic heart failure is caused by vagal neuritis, since there is often a neuritis of the spinal nerves. The onset of cardiac failure in this stage is often sudden with abdominal pain and vomiting and death following in a matter of hours or days. The heart is often enormously dilated, probably the result of a loss of tonus in the myocardium. There is an intracardial tonus center near the sinus node and the dilatation may be a consequence of the poisoning of this center. Although the oxygen consumption in such a dilated heart is about three times that of a normal heart, the vascular atony prevents an increase in blood supply and sudden death is the result.

It is seen therefore that the failure of the vascular system is important in the heart failure of all three stages of diphtheria. This circulatory failure is not well understood but is probably closely connected with other shock-like conditions; the peripheral vascular system, the autonomic nervous system, and the cortex of the adrenals all being involved.

From the point of view of therapy, the most efficient treatment is the application of heat. The patient is put in a hot bath in which the temperature is slowly raised from 35 degrees centigrade to 39 degrees centigrade. If restlessness becomes evident, the treatment is interrupted. Dr. Friedemann has found this method very effective in the first and third stage circulatory failure but of no use in the second stage.

BOSTON MEDICAL HISTORY CLUB

The Boston Medical History Club met Monday evening, December 17, in Sprague Hall of the Boston Medical Library. Dr. Henry R. Viets opened the meeting and commented on the recent death in Paris of Stephen d'Irsay, an authority on medical history, whose latest publication was "Histoire des Universités."

Mr. James F. Ballard presented an impressive array of books and manuscripts written between 1250 and 1500 belonging to the Boston Medical Library. Mr. Ballard discussed each book in turn, commenting on the author, the contents, and the nature of the material on which the book was written or printed. The early manuscripts were copied by monks, and vellum was used exclusively. A little later paper began to replace the vellum, and with the advent of the printing press after the middle of the fifteenth century, books began to be produced in quantities. Toward the end of this period wood-

cuts began to appear, and many of the publications owe their fame to some particular illustration.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, January 15, 1935, 12 noon. The speaker will be Louis M. Spear, M.D., Physician-in-Chief, Robert B. Brigham Hospital. His subject will be "Chronic Arthritis." All physicians are invited to attend both lecture and luncheon.

NEW ENGLAND PHYSICAL THERAPY SOCIETY

Evans Memorial Auditorium, 80 East Concord Street, Boston, January 16, 1935, at 8 P.M.

Second Lecture on Electricity

By Dr. L. L. Campbell

A. Some Effects Produced by Electric Currents.
   1. Thermal effects due to direct currents.
   2. Thermal effects due to alternating currents.
   3. Chemical effects: electrolysis; iontophoresis; cataphoresis.
   4. Electromagnetic effects: motor-generator; transformer; condenser; spark-gap; etc.

B. The Physics of Some Electrotherapy Apparatus.
   1. Apparatus for galvanism; direct and sinusoidal currents.
   2. Diathermy apparatus; the electrode and the inductotherm types.

Physicians and medical students are cordially invited to attend.

ARTHUR H. RING, M.D., *Secretary.*

Arlington.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance) Tuesday evening, January 8 at 8:15 P.M.

PROGRAM

"Forty Years of Diabetes" with Presentation of Cases. By Dr. Elliott P. Joslin.

MARSHALL N. FULTON, M.D., *Secretary.*

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society will be held January 11 in the Auditorium of the Beth Israel Hospital, Boston, at 8:00 P.M.

PROGRAM

Speaker: Dr. Charles F. McKhann, Children's Hospital.

Subject: "Immune Substances in Placental Extract."

Chairman: Dr. Edwin H. Place, Professor of Clinical Pediatrics, Tufts College Medical School.

CLINIC AT THE CHILDREN'S HOSPITAL

A clinic at which orthopedic, medical and surgical cases are to be presented will be held on Monday, January 7, at 4:00 P.M. in the Amphitheatre of the Children's Hospital, Boston.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JANUARY 7, 1935

Monday, January 7—

9 A.M. Boston Surgical Society, New England Deaconess Hospital.

†4 P.M. Pediatric Clinic. Children's Hospital Amphitheatre.

8:15 P.M. New England Heart Association. Peter Bent Brigham Hospital Amphitheatre.

Tuesday, January 8—

†2:30-4 P.M. Ward Visit (Pediatrics), Massachusetts Eye and Ear Infirmary.

†4-5 P.M. Seminar Pediatrics, Massachusetts General Hospital, Pediatric Laboratory.

*8:15 P.M. Harvard Medical Society, Peter Bent Brigham Hospital Amphitheatre.

Thursday, January 10—

*12 M. Clinico-Pathological Conference, Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference, Children's Hospital.

*3:30 P.M. Medical Clinic. Dr. Christian, Peter Bent Brigham Hospital.

†4:30 P.M. Surgical Clinic. Peter Bent Brigham Hospital.

Friday, January 11—

8 P.M. William Harvey Society, Beth Israel Hospital.

Saturday, January 12—

*10-12. Medical Staff Rounds, Dr. Christian, Peter Bent Brigham Hospital.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

January 7—New England Heart Association will meet at the Peter Bent Brigham Hospital at 8:15 P.M. For details address the Secretary, Dr. James M. Faulkner, 264 Beacon Street, Boston, Mass.

January 7—Clinic at the Children's Hospital. See notice above.

January 8—Harvard Medical Society. See notice elsewhere on this page.

MASSACHUSETTS DIETETIC ASSOCIATION

January 8—Tuesday, 8 P.M. "Careers and Natural Abilities," Professor Johnson O'Connor, Human Engineering Laboratory, Stevens Institute of Technology, Hoboken, N. J.

February 12—Tuesday, 8 P.M. "Diabetic Children," Dr. Priscilla White, Joslin Diabetic Unit.

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

January 10—Clinic at the Peter Bent Brigham Hospital. See page 35.

January 11—William Harvey Society. See notice elsewhere on this page.

January 14 - February 25—Lectures and Clinics on Heart Disease by Dr. Christian. See page 35.

January 15—South End Medical Club. See notice elsewhere on this page.

January 16—New England Physical Therapy Society. See notice elsewhere on this page.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Semi-Annual Meeting will be held in January and the Annual Meeting in May, time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of January, March, and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 9, 1935—Melrose.
March 13, 1935—Wakefield.
May 8, 1935—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January—Goddard Hospital.
March—Plymouth County Hospital.
April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 23, 1935—General Meeting in association with the Boston Medical Library. Speaker and subject to be announced later.

March 27, 1935—Clinical Meeting at the Boston Lying-In Hospital.

April 24, 1935—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.
HENRY T. HUTCHINS, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

January 9, 1935—Wednesday evening. Worcester City Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Buffet supper complimentary by the Hospital.

February 13, 1935—Wednesday evening. Worcester State Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

March 13, 1935—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10, 1935—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8, 1935—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

To Remind. A Biological Essay. The Abraham Flexner Lectures. Series Number Two. By Sir William Bate Hardy. 45 pp. Baltimore: The Williams & Wilkins Company.

The Principles of Therapeutics. The Abraham Flexner Lectures. Series Number Three. By Francis Richard Fraser. 135 pp. Baltimore: The Williams & Wilkins Company.

Minor Surgery in General Practice. By W. Travis Gibb. 429 pp. New York: Paul B. Hoeber, Inc. $5.00.

Marriage and Sexual Harmony. By Oliver M. Butterfield. 40 pp. New York: Emerson Books, Inc. $.50.

Clinical Pathology of the Jaws. With a Histologic and Roentgen Study of Practical Cases. By Kurt H. Thoma. 643 pp. Baltimore and Springfield: Charles C. Thomas. $9.00.

Mental Health, Past, Present, and Future. The Colver Lectures, 1932. By Arthur Hiler Ruggles. 104 pp. Baltimore: The Williams & Wilkins Company.

Definite Diagnosis in General Practice. By W. L. Kitchens. 1000 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

A Textbook of Pathology for Nurses. By Coleman B. Rabin. 243 pp. with 61 illustrations. Philadelphia and London: W. B. Saunders Company. $1.75.

Manual of Clinical Laboratory Methods. By Pauline S. Dimmitt. 156 pp. Illustrated. Philadelphia: F. A. Davis Company. $2.00.

The Heart Visible. By J. Polevski. 207 pp. Philadelphia: F. A. Davis Company. $5.00.

The Equilibrated Salt Diet. By Robert Wollheim and Walter H. Schauinsland. 64 pp. New York: Professional Scientific Service.

Osteomyelitis. Its Pathogenesis, Symptomatology and Treatment. By Abraham O. Wilensky. 454 pp. New York: The Macmillan Company. $9.00.

Medical Art Calendar 1935. J. Philip Kruseman. The Hague. $1.75.

Traité Elémentaire D'Exploration Clinique Médicale. (Technique et Séméiologie.) Par Émile Sergent et al. 1176 pp. Paris: Masson et Cie. Broché 120 fr., Cartonné toile 145 fr.

Gynecology. By Brooke M. Anspach. Fifth Edition. With the assistance of Philip F. Williams and Lewis C. Scheffey. 832 pp. Philadelphia and London: J. B. Lippincott Company. $9.00.

BOOK REVIEWS

Institutional Care of Mental Patients in the United States. By John Maurice Grimes. 138 pp. Privately published. Chicago, 1934. Price $3.00.

This book consists of a privately printed report which grew out of an investigation made by the author for the American Medical Association. This investigation was started by a resolution passed in June, 1930 with the idea of surveying all hospitals caring for mental patients in the United States, both public and private. The author, with one or two colleagues, carried out this work systematically, partly by the method of questionnaires and partly by personal visits. Of the 631 institutions considered, seventy-five per cent of the superintendents responded to the questionnaires and some member of the investigating board made personal visits to 600 of the hospitals. The expenses of this work were defrayed by appropriations from the American Medical Association. A brief preliminary report was given in 1933, but the author states that no extended report was ever made and the results of the investi-

gation were partially suppressed. He now has taken it upon himself to give the report in detail and send it out to various interested persons as a private publication.

The report is divided into three parts, the first concerning publicly controlled institutions. Of these there are 251 state institutions, 174 of which are for the insane and 65 for the mentally deficient. The others are for epileptics and drug addicts. Many details are given of these hospitals, both in regard to their location, their equipment, the medical personnel, technical staff, social service, etc. In general, the author feels that the medical personnel of most state hospitals is inadequate and the good men are always too few and too busy. For the most part, the federal institutions are newer and much better equipped and provided for financially than the older state institutions. The second part of the report deals with private hospitals. The author found these on a relatively high plane and most of them could justly be called hospitals, where definite therapy was given and in which the primary function was not merely custodial care. The third part of the book takes up special considerations, such as social and medical care. He believes that most of the state hospitals lack an adequate staff of social workers, that the medical care is so curtailed that in most instances it is unfair to speak of the state institutions as hospitals, and finally, that the financial support is so meagre that honest effort by the physicians cannot be given under existing circumstances. After this thorough study, the author feels that the solution of the mental patient problem is not merely additional money, but that more adequate care, and particularly better medical attention, should reduce the number of patients in institutions to such an extent that the financial burden of caring for them should be even less than it is to-day. The whole medical staff should be concentrated on the problem of de-institutionalization, with the definite aim of paroling all parolable patients. Secondly, the parole service should be under competent medical direction, and thirdly, more definite "acute" service should be arranged for in these hospitals so that the last vestiges of the old insane asylum should disappear. The final step will be the redisposition of the "chronic" service, under economic and sociological direction with medical supervision. The book is completed by a very carefully compiled table of all the hospitals in this country and a number of useful maps.

Although one cannot comment upon the controversy which the author has had with the American Medical Association, one feels that this report is of considerable value. The facts appear to be accurately given and the deductions broadminded and sane. To all physicians and others who have under their care the half-million cases of mental disease in this country now in institutions, this book is recommended as one that should lead to a reconsideration of the whole problem. The personal bias of the author has only slightly interfered with the worth of his publication.

Tumors of the Female Pelvic Organs. By Joe Vincent Meigs, with a foreword by Robert B. Greenough. 261 illustrations. 533 pp. New York: The Macmillan Company. $6.00.

It is well that we have correlated and brought to our attention more frequently, certain pathological groups with their clinical manifestations and the results of our present methods of treatment. Too often we have the pathology of a group of cases most thoroughly presented, with but scanty mention of the clinical and therapeutic aspects—and, on the other hand, clinical details and methods of treatment with merely the names of the pathological material treated. It is a pleasure to find a book, such as Dr. Meigs has just written, covering a specific pathological group and telling in clear language what the clinical manifestations, treatments and results are for this particular group.

"Tumors of the Female Pelvic Organs" is, as is stated in the preface, a survey and as such, is of great value. It necessarily includes many statistics, both of cases from the wealth of material from the Massachusetts General Hospital and from other clinics. These statistics are so clearly presented that there is no confusion and one can easily find out what is the consensus concerning treatment. This is of real value.

The illustrations, which are abundant, are most excellent, especially the photomicrographs. The chapter on metastases is valuable. The text is clear and all viewpoints in regard to treatment are presented and the results of each summarized and evaluated. For the student and the surgeon who wish to know the present status of the pathology and treatment of pelvic tumors, no clearer presentation could be asked for.

Conception Period of Women. By Kyusaku Ogino. 95 pp. Harrisburg, Pa., Medical Arts Publishing Company. $1.00.

Since the war there have been great advances in our knowledge of physiology and reproduction. It is during this same period that there has been a great increase in the interest shown in contraceptive methods and technique. Our ideas of the physiology of conception have undergone merely as great changes as have our ideas in regard to such other branches of this subject as the importance of the hormones, or the physiology of menstruation. Within the last year or two we have been hearing a good deal of a new contraceptive method based upon this newer knowledge of the female reproductive physiology. Popular books have appeared, notably "The Rhythm" by Leo J. Latz, M.D. There has been a great deal of assertion without very much of fact to substantiate it. With the appearance of a new popular book "Conception Period of Women" by Dr.

Kyusaku Ogino it seemed wise to find out what real scientific evidence could be found for the claims which have been made for the new method.

The method as advocated by Ogino is in brief as follows: In the first place the method is useful only for women whose menstrual cycles are quite regular. Provided that a woman has found this to be the case she is to count back on her calendar eleven days from the date of her expected menstruation. These eleven days constitute the so-called safe period. Preceding them there is a dangerous period of eight days and preceding this a relatively short stretch of time to the end of menstruation. This last relatively safe period may be obliterated if the cycle is less than twenty-one days or of considerable duration if the cycle is more than twenty-eight days. The invariable rule is that all periods must be reckoned not in days after the beginning of menstruation but in days preceding an expected one. The dangerous eight days are arrived at by allowing for some variation in the time of ovulation for the individual woman, for the life of the egg which probably does not exceed one or two days, and for the length of time during which spermatozoa retain their ability to fertilize, probably not more than three days.

Ogino's book consists of seven chapters, a bibliography which is very complete and an appendix consisting of a reprint of an article appearing in the June, 1933 issue of *Surgery, Gynecology and Obstetrics* on this same subject by Dr. A. G. Miller and his associates of the Miller Clinic in Hobart, Indiana. The appendix, the chapter on the period of ovulation, and those on the life of the ovum and the life of the spermatozoa give a very comprehensive review of all the scientific evidence bearing on these subjects. Chapter V brings forth the author's arguments bearing on the period of human conception. The last three chapters deal with the practical application of this theory and are written in a more popular vein.

The original German papers of Siegel and Nürnberger who reported on the conception time of German women during the war seemed to show a period of maximum fertility for the whole week following menstruation which was succeeded by a period of relative sterility until the next menstruation appeared. Capellmann and Niedermeyer based their theories upon this work but a careful appraisal of it would indicate that the statistics were very carelessly gathered and are not at all reliable. Ogino quotes papers of his own appearing in Japanese publications as early as 1923. His first article in German was in 1930. Before that time Knaus published an important article in the *Zentralblatt für Gynecologie* in 1929 on a new method for determining the end of ovulation. This paper reviewed his previous work with animals and reported thirty-six cases in which the method had been tried with human beings. As a result he was able to say that ovulation occurred during the dangerous period noted above and to base on this a theory of conception control. These theories of Ogino and Knaus have been widely circulated on the Continent particularly by a Dutchman named Smulders. The latter has published a book based on the so-called law of Ogino-Knaus. In this country scattered articles have appeared, the most scientific being that of Miller already referred to and there is the book of Latz which is an entirely popular presentation and appears with ecclesiastical approbation.

After reading Ogino, Knaus and the other sources as published in the German and after checking all the original evidence adduced by Ogino from operations, personal cases, etc., one cannot help being impressed with the reasonableness of the theory, one might almost say the probability of its correctness. But when one looks for controlled series of human statistics one cannot help being disappointed. To date the article by Miller covering data regarding eighty-seven couples with some 700 copulations and the cases reported by Ogino of six couples using this method for an aggregate of 142 months and 300 copulations, together with a few scattered reports in the literature are about all we have to go on. Ogino claims after collecting these scattered cases and the ones mentioned above to have found 500 cases for his theory and very few against it.

The situation then at the present time may be summarized as being still in the stage of theory. It is true that this theory coincides with our present knowledge of physiology much more completely than the one which makes conception possible at any time in the menstrual cycle, but it is also true that before giving advice to medically sick patients in whom pregnancy would be dangerous to life, or even somewhat harmful to health, we have got to have a great deal more carefully controlled evidence from human matings than is at present available.

Developmental Anatomy. A Text-Book and Laboratory Manual of Embryology. By Leslie Brainerd Arey. Third Edition, Revised. With 547 illustrations, many in colors. 593 pp. Philadelphia and London: W. B. Saunders Company. $6.50.

This new edition of a familiar textbook on embryology comes just four years after the publication of the last one. Because of its inclusiveness, a review of the literature with a corresponding revision of the former text represents a very considerable task for the author.

Those who have used a previous edition as a reference or study book will remember how well it combined scientific accuracy with a lucid and interesting style. One has only to read the chapters on the complicated development of the urogenital organs to see how expertly Dr. Arey handles his materials, using text with diagrams to unfold a logical sequence in development. A very fine feature is a table giving the stages of development of practically all the parts of the body at different periods; and the student of embryology will especially appreciate

the superscripts directing the reader to original sources. Men in every field of medicine will find this a handy reference when particular facts of embryology are desired.

This edition, in common with those previously published, contains a laboratory manual. The workmanship of the book is excellent.

Obstetric Medicine. Edited by Fred L. Adair and Edward J. Stieglitz. 743 pp. Philadelphia: Lea and Febiger. $8.00.

This book is a series of articles by 39 contributors, edited by Drs. Adair and Stieglitz. In their introduction the editors state that they have chosen only the more common and important of the medical conditions for presentation, and that they tried to impress upon the contributors the idea that each chapter should be concise, definite, and should not be a theoretical exposition of the subject.

In the first section, under "General Considerations," the physiology of normal pregnancy, physical diagnosis in pregnancy, pharmacology and fetal disturbances are covered interestingly and well.

Section II includes the "Specific Infectious Diseases." The chapter on "Tuberculosis" is excellent, and it ends with a full bibliography. The chapter on "Syphilis" is as the writer describes the disease, "a florid generalization". It lacks specific details of treatment, and there is no bibliography. The chapter on "Venereal Diseases" is excellent, full of specific treatment and conservative advice. That on "Contagious Diseases" is short and contains nothing that is of great help in their relation to obstetrics. The chapter on "Tropical Diseases" and that on "Parasitic Diseases" are interesting.

Section III contains chapters on "Non-Infectious Diseases," the outstanding one being that on "Deficiency Diseases."

Section IV is on "Disorders of the Nervous System" and Section V is on "Disorders of the Respiratory Tract."

Section VI takes up "Disorders of the Circulatory System," and under "Diseases of the Heart" the various types of cardiac disturbances are carefully discussed. The section does not leave the reader with a clear idea of the treatment and management of obstetric cases with heart complications.

In Section VII "Disorders of the Alimentary Tract" are taken up, and there is a routine discussion of the various conditions and complications that come in these diseases.

Following this are sections on "Disorders of the Urinary System," "Endocrine Balance," "Blood," "Body Mechanics," and the book ends with a chapter on "Diseases of the Skin."

They are all satisfactory articles on the diseases studied. They vary greatly in their bearing on obstetrics. Much in the chapters could be entirely eliminated and left to books on medicine. In some of these chapters there is no bibliography, and the treatment suggested is not specific. As is to be expected in a book with so many contributors, there are many statements that do not coincide. Some are completely contradictory. The book contains a mass of interesting material. It does, however, lack specific details in the treatment of many conditions considered, which the editors lead us to suppose would be given.

The Surgical Clinics of North America. August, 1934. Volume 14—Number 4. Chicago Number. 288 pp. Philadelphia and London: W. B. Saunders Company. Per clinic year February, 1934 to December, 1934. Paper, $12.00; Cloth, $16.00.

This issue opens with an excellent symposium on plastic surgery, an important subject that has received relatively little recent attention. Moorehead illustrates the value of elastic traction to close rather large defects by the use of adjacent tissues. This simple principle is worthy of much more general use. Koch describes the various methods available for the correction of burn contractures of the axilla, more particularly the Z incision, pedunculated flaps, and split grafts. C. and W. C. Beck report the successful grafting of toes to fingers and also describe their operation of plastic correction of the pendulous breast. J. C. Beck and Guttman report a case illustrating the use of the tube graft to cover a large facial defect. An unusual type of plastic repair of hypospadias is described by Gatewood. He makes a circular "umbrella" flap about the opening, inverts it, and transplants it distally.

Friends of the late Dr. Carl A. Hedblom will be interested in his clinic given with Van Hazel on the surgical treatment of tuberculosis. His cases illustrate apical thoracoplasty for residual cavity, extrapleural thoracoplasty for tuberculous empyema, inadequate pneumothorax with recurrent effusion treated by posterior thoracoplasty with upper-stage anterolateral resection, extensive chronic tuberculosis with dextrocardia with satisfactory result following a three-stage extrapleural thoracoplasty, and open thoracoplasty for adhesions followed by partial extrapleural thoracoplasty.

Another noteworthy group of clinics is in the nature of a symposium on peptic ulcer with Bevan discussing the surgical aspects, Brown the medical treatment, and Rose the roentgenological features.

These with many other valuable contributions make this an outstanding number.

Postures and Practices During Labor Among Primitive Peoples. By Julius Jarcho. 175 pp. With 130 Illustrations. New York: Paul B. Hoeber, Inc. $3.50.

The author has obviously spent much time investigating the curious and terrible customs in obstetrics that have prevailed in various countries in ancient and modern times. It is distinctly worth while to have an account of these customs grouped in one book where they are readily available to the student of obstetrical history.